NERVE INJURIES AND SHOCK

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R.A.M.C. (T.)

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NERVE INJURIES AND SHOCK

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NERVE INJURIES AND SHOCK

BY

WILFRED HARRIS

M.D. (Cantab.), F.R.C.P. (Lond.)

PHYSICIAN TO ST. MARY'S HOSPITAL, LONDON, AND TO THE HOSPITAL
FOR EPILEPSY AND PARALYSIS, MAIDA VALE,
CAPTAIN R.A.M.C. (T.), 3RD LONDON GENERAL HOSPITAL

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PREFACE

An outstanding feature of the casualties in the present war is the very large number of cases of nervous exhaustion, neurasthenia, and functional paralyses of various kinds which are being met with.

Many occur as the result of large shell explosions, burying the man in the blown-in parapet of a trench, or hurling him down, without any local injury, yet causing temporary unconsciousness, and leaving behind a flaccid paraplegia, or other symptoms characteristic of loss of function from nervous shock. Neglecting the Greek derivation of the word, the term hysterical paralysis may be used to describe many of these cases, instead of the wider and vaguer term functional paralysis, and this condition, with or without hysterical contracture and anæsthesia, may be superimposed upon an actual nerve injury, rendering the clinical picture bizarre and difficult to unravel to one off his guard or unfamiliar with these cases.

With proper methods of suggestive treatment, these cases of nervous shock can nearly all be rapidly, or even immediately cured, and in this little book I have endeavoured to give as briefly as possible the important features of the various forms of nervous injury met with, and the appropriate treatment, in the hope that it may be of service to others interested in nervous diseases.

WILFRED HARRIS.

56 *Wimpole Street, W.*
August 24, 1915.

CONTENTS

PART I

DIRECT EFFECTS OF INJURIES OF THE NERVOUS SYSTEM

CHAPTER I

INJURIES OF THE BRAIN AND THE CRANIAL NERVES

PAGE

Cranial Nerve Paralyses. Concussion. Compression. Contusion. Jacksonian epilepsy. Contrecoup, Treatment of cerebral injuries. After-treatment. Remote effects. Facial neuralgia. Cerebral abscess 9

CHAPTER II

INJURIES OF THE SPINAL CORD

Spinal concussion. Intrameningeal spinal hæmorrhage. Fractures and fracture-dislocations of the spinal column. Total transverse lesions. Remote effects. Pain in the back. Lesions of cauda equina . . 31

CHAPTER III

INJURIES OF THE BRACHIAL PLEXUS

PAGE

Cervical rib. Paralysis of the trapezius. Posterior scapular nerve. Paralysis of the serratus magnus. Paralysis of the posterior cord. Paralysis of the outer cord. Paralysis of the inner cord. Dislocation of the shoulder. Musculo-spiral paralysis. Median nerve paralysis. Occupation neuritis. Ulnar nerve paralysis. Ascending neuritis. Brachial neuritis. Painful stump. Ischæmic myositis and neuritis. Traumatic neuro-fibrositis . . . 53

CHAPTER IV

INJURIES OF THE LOWER EXTREMITY

Anterior crural nerve injury. Obturator nerve. Sciatic paralysis. Sciatica 79

PART II

NERVOUS SHOCK FOLLOWING CEREBRAL INJURY

General paralysis of the insane. Neurasthenia. Treatment of neurasthenia. Nervous exhaustion. Hysteria. Hysterical monoplegia. Treatment of hysterical paralysis and anæsthesia . . . 92

INDEX 125

PART I

NERVE INJURIES

CHAPTER I

INJURIES OF THE BRAIN AND THE CRANIAL NERVES

INJURIES to the brain as the result of violence will vary in extent and character according to the weapon and the force of the blow; thus injuries may be inflicted by violent contact with (1) a hard blunt instrument, such as a paving stone, or against a wall; (2) by a heavy semi-solid vessel, such as a sandbag (an American hooligan method of attack); (3) or the wound may fracture the skull, as by a bullet, dagger, or bayonet, pickaxe, or sharp fragment of stone or steel. Speaking roughly, the severity of the injury will be less severe in the case of the semi-solid sandbag, and most dangerous from the penetrating wound.

One of the commonest injuries is that resulting from a violent fall on the head upon a hard pavement. In this case there may or may not be local injury to the scalp to be seen, such as a hæmatoma, bruise, or cut. When great violence has been used there may be a depressed fracture visible or palpable at the site of the injury, though in some cases, especially if not examined immediately after the injury, the hæmatoma or œdematous swelling may obscure the depression, which may consequently be overlooked. With or without a local depressed fracture, fracture of the base of the skull often occurs as the result of such violence, sometimes more than one line of fracture traversing the skull in various directions.

Cranial Nerve Paralyses

A not uncommon course for the line of fracture is to traverse the petrous bone, causing rupture of the tympanum, with consequent hæmorrhage from the artery supplying it, and free bleeding may result, which may be entirely external if the patient happens to be left lying with the head on that side for some time after the accident ; in other positions the blood may trickle down the Eustachian tube into the œsophagus and stomach, giving rise to subsequent hæmatemesis and melæna. The line of the fracture may involve certain cranial nerves at the base, particularly the seventh and eighth nerves, causing facial paralysis and nerve deafness from injury to the petrous bone, or one or other of the nerves supplying the eye muscles may be involved, the third, fourth, and sixth cranial nerves, causing various forms of diplopia, visible squint, or ptosis. Not infrequently a single muscle may be involved, such as the external rectus, from sixth-nerve palsy, causing convergent squint and homonymous diplopia, or a single branch of the third nerve may be paralysed, such as the branch to the inferior oblique, in which case no squint will probably be visible, but homonymous diplopia will be present, most marked on looking outwards and upwards to the affected side.

Occasionally the fifth nerve will be paralysed as the result of a violent blow on the head, probably due to the line of the fracture involving the Gasserian ganglion. I have seen only two instances of this, in both of which cases the fifth-nerve paralysis was permanent. On the other hand, diplopia as the result of the injuries involving the nerves to the eye muscles may clear up after some weeks or months, though this symptom also may be permanent, as also may be the facial paralysis and nerve deafness of similar origin. Injury to the third nerve, causing divergent squint, crossed diplopia, and ptosis, with sluggish and semi-dilated pupil, may be completely recovered from, with the exception of the paralysis of the pupil. For instance, I have seen typical Argyll-Robertson pupil on one side, the pupil being medium contracted, with no reaction whatever to light, but brisk contraction to convergence and accommodation, as the only sequel remaining from such a lesion. Injury to the third nerve from fractured base in childhood may leave as a sequel the curious condition known as *ophthalmoplegic migraine*, in which there is permanent partial paresis of the third nerve, but at wide intervals of many months, or even years, the patient is taken acutely ill with severe headache, and the third nerve becomes more or less completely paralysed for several days, gradually recovering during the next two or three weeks. The line of the fracture also may pass through the centre of the sphenoid and may involve the optic chiasma, and I have seen bi-temporal hemianopia more than once produced by this lesion, thus damaging the decussating fibres in the chiasma, which take their origin from the nasal halves of the two retinæ. The temporal fibres which supply the nasal fields of vision do not decussate at the chiasma, but pass round its outer borders into the optic tracts of the same side, and are therefore less liable to injury.

Bi-temporal hemianopia is characteristic of a lesion at the chiasma, such as may result from sudden injury, or from gradual pressure from above by a pituitary tumour, or by a dilated third ventricle. Wernicke's hemianopic pupil sign may also be obtained in such a case, if carefully tested for, in which a fine parallel pencil of light is focused on the pupil, first from one side and then from the other. When the light is thrown upon the blind half of the retina no pupil reaction takes place, though this reacts subsequently to light coming from the opposite direction. This sign will differentiate hemianopia due to a lesion of the optic tract or chiasma from hemianopia due to an intracranial lesion of the optic radiations, or of the half vision centre in the neighbourhood of the calcarine fissure on the inner aspect of the occipital cortex. The optic nerve itself may suffer from the line of fracture involving the optic foramen in the sphenoid, or both optic nerves may be cut through at this level by a bullet wound. As the result of such an injury, complete and immediate blindness of one or of both eyes will ensue, and subsequently simple white atrophy of the optic disk will develop. The reason for this is not clear, as the trophic cells for the optic nerve-fibres lie in the retina, the degeneration thus apparently ascending, as well as descending in the opposite direction as far as the external geniculate ganglion and the anterior corpus quadrigeminum. Fracture of the base not infrequently involves the cribriform plate of the ethmoid, causing unilateral or bilateral loss of sense of smell; this is usually permanent.

Thus the first eight cranial nerves are liable to injury from fractured base, the bulbar nerves from the glosso-pharyngeal to the hypoglossal being rarely involved.

Concussion of the Brain

In the less serious forms of cerebral injury the patient may not lose consciousness, though numerous symptoms may be present, on careful observation, which will indicate a cerebral disturbance, or commotio cerebri, usually known as concussion. The pathological processes underlying these conditions are not known with certainty, chiefly for the reason that the milder forms of concussion are rarely fatal, though it has been suggested that fine hæmorrhages are likely to be found in the meninges and cortex, in addition to more diffuse and microscopic changes, such as cerebral œdema and nutritive changes in the nerve-cells of the cortex. It is more than doubtful, however, whether hæmorrhages or any recognizable macroscopic or microscopic lesion are necessarily present, as occasionally a fatal result ensues after cerebral injury and concussion in which no lesions of the brain or meninges have been found.

The patient who has been concussed will have probably vomited, the face and lips will be pallid, the skin cold and clammy, pulse often altered, either slow or rapid, and complaining of headache, with perhaps pain and stiffness of the neck. The pupils are often dilated and perhaps show irregular alterations in size, and do not remain firmly contracted under the stimulus of light. Such a patient may be able to answer questions, though his answers are not necessarily relevant, and he may be even garrulous, while in other cases answers are slowly evoked, the cerebral processes of thought apparently requiring a very long latent period. Memory of this period is always confused after the patient has recovered, and very probably there may be a complete blank both for the incidents shortly preceding the accident as well as for varying intervals following the injury.

In different cases varying degrees of loss of consciousness may be observed, down to profound coma, with loss of corneal reflex and pupil reflex to light, with snoring, irregular respiration, and complete incontinence. Again, occasionally the onset of serious symptoms may be delayed, and a patient who two or three hours after the accident may still be conscious and able to walk, though probably exhibiting the signs of concussion above enumerated, may later relapse gradually into profound unconsciousness and coma.

Cerebral Compression

Ingravescent symptoms usually indicate cerebral compression, which may be due either to the development of œdema of the brain from the vasomotor commotion set up by the injury, or it may be due to compression of the brain by slowly increasing hæmorrhage, which may be either extra-dural, as from a ruptured meningeal artery or one of its branches, or from hæmorrhage on the surface of the cortex from a ruptured vein or artery in the pia-arachnoid, or in a few cases by subcortical hæmorrhage from a vessel ruptured within the cerebral substance. It will be impossible clinically to distinguish these varying causes of cerebral compression, the general symptoms of compression being closely similar in each case, though varying with the part of the brain which is affected. Cerebral compression from one of these four causes, œdema of the brain, intracerebral hæmorrhage, meningeal hæmorrhage, or extra-meningeal hæmorrhage, may sometimes be distinguished from compression due to a depressed fracture by the fact that the coma in depressed fracture is initial and prolonged, whereas, in the former group of causes, coma is sometimes delayed in its onset, or the patient, unconscious at first from the concussion, may recover consciousness, only to relapse again into unconsciousness and coma with increasing symptoms of compression. The pulse, at first accelerated, becomes slow and irregular, and harder to the finger, the respiration also failing in frequency and becoming irregular, or even Cheyne-Stokes in character; the pupil on the affected side, at first slightly dilated, will become more contracted than the opposite pupil during the earlier stages of the compression, again becoming dilated when the coma is profound and the respiration is failing and a fatal end is near.

As soon as the onset of cerebral compression is recognized, it is important that the operation of trephining the skull should be performed without delay, and a sufficiently large skin-flap should be made in order that a large area of bone may, if necessary, be removed. Hæmorrhage from the middle meningeal artery is usually easily recognized and dealt with; but in cases where compression is due to subcortical hæmorrhage, or to cerebral œdema, the cause may be overlooked when, through a comparatively small trephine opening, it is seen that there is no meningeal hæmorrhage, and the mistake may be made of closing the wound without doing anything further. A larger opening in the bone must be made, and if compression is present, the cortex will at once begin to push out through the opening, thus relieving the tension, and pulsation of the brain will then reappear. Quite a large bony opening, 3–3½ inches by 2 inches, may be necessary. In some cases of subcortical hæmorrhage the clot may be indicated almost presenting at the surface, and a small incision in the cortex may succeed in evacuating the clot, when the bone may be replaced with advantage.

Cerebral Contusion

After severe blows on the head, laceration of the cortex, with hæmorrhages both into the cortex and into the meninges, is a not uncommon injury. In these cases coma is immediate and likely to be much more prolonged than in an ordinary concussion, and the temperature begins to rise after the first twenty-four hours, fluctuating irregularly perhaps for weeks, while delirium is apt to follow the coma after a day or two, the patient perhaps muttering and talking continually, and resenting examination. At times he may be violent and shrieking, perhaps maniacal; at other times he may appear to understand some questions and to recognize certain people. There is complete incontinence of the sphincters during this period, and insomnia is a pronounced feature, though in many cases he can be got to take fluid nourishment fairly well. Usually there are no local signs of cerebral injury detectable at this time, such as may be present in cases of cerebral compression, as hemiplegia, or paralysis of one limb, or of one side of the face. The pulse will usually be rapid and regular, the respiration slightly increased, while the absence of the signs of compression will help to distinguish contusion from the latter condition. Lumbar puncture too may be of service, as a clear colourless fluid will indicate the absence of any intrameningeal hæmorrhage, whereas if such has taken place to any degree, the fluid obtained by lumbar puncture will be uniformly stained reddish during the first three days. After the fourth day the fluid assumes a yellow tint from alterations in the blood pigments, and then does not give the guaiac test for blood pigment.

The course of these cases is a very anxious one for the friends, and may be prolonged to six or eight weeks or even longer, before the delirium ceases and consciousness commences to be regained. Favourable symptoms will be control of the delirium by means of bromide and chloral compounds, and the ability to take and retain nourishment. The after history of these cases will depend considerably on the skill in nursing and upon the duration of the rest insisted on. This must be prolonged, and all influences for excitement must be carefully withheld, and the patient encouraged to sleep and to avoid talking, and to rest his brain in every possible way for several months. Exercise similarly must be very little indulged in for the first few months after recovery. Symptoms of focal lesion sometimes appear after cerebral contusion, and though not apparent during the early stages of coma and delirium, they may become detectable after the regaining of consciousness. Thus, laceration of the cortex may set up adhesions and scar formations between the meninges and cortex, which will give rise to focal symptoms of paralysis if the motor or precentral area is involved. Weakness and clumsiness of one hand or foot or of the entire leg may thus be complained of, with increase of the knee and Achilles jerks, or of the arm reflexes, with an extensor plantar reflex and absence or diminution of the abdominal reflexes.

Aphasic symptoms of varying character and degree may be present from lesions in the neighbourhood of the posterior portion of the left second frontal, or of the superior temporo-sphenoidal convolution. Lesions of the occipital pole may give rise to partial hemianopia, involving both the upper and lower half fields of the opposite side, or only the upper or lower quadrants of the visual field on the opposite side for each eye, according as to whether the lesion involves the cortex below and adjacent to, or above the calcarine fissure respectively. Lesions of the parietal cortex often may give rise to no definite signs, though extensive slight hemianæsthesia of the opposite side may be noticed, and if damage involves the parietal convolution just posterior to the fissure of Rolando, convulsive attacks may ensue, closely resembling the focal spasms of Jacksonian epilepsy, which are a frequent sequel of focal lesions in the pre-Rolandic or motor cortex.

Jacksonian Epilepsy

Unilateral convulsions are one of the commonest results of cerebral injury, and may occur almost at any time after the accident. One or two such attacks may occur half an hour or more after a slight concussion, being accompanied by unconsciousness, which is gradually recovered from during the next few hours. Such attacks do not necessarily mean any grave injury, and the patient may completely recover without any trace of cerebral damage remaining. It is most important therefore in the early treatment of cerebral injury to avoid operative interference, unless such interference is pressingly called for by symptoms of compression from depressed fracture, cerebral œdema, or hæmorrhage (see p. 15). On the other hand, recurrent Jacksonian convulsions may occur under like circumstances, the patient becoming comatose some few hours after the accident, with delirium between the bouts of convulsions, and death may supervene without any recognizable lesion of the brain or of the meninges to be found post mortem. Operation in such cases by trephining undoubtedly hastens the fatal ending, and should never be undertaken unless clear signs of compression be present. Indeed it is probable that many deaths have been hastened through such operative interference in patients who might have recovered if they had been left alone. In another category are the Jacksonian convulsions which occur in cases of cerebral contusion after recovery from the accident. These may be frequently repeated, leading to considerable interference or total interruption of the patient's ordinary life, and to considerable clouding of the intelligence and mental hebetude owing to the repeatedly recurring convulsions. In such a case the question of trephining and excision of the irritant focus in the cortex must be carefully considered, and the operation may lead to brilliant results if carefully performed, after the case has been previously thoroughly studied. Thus, careful note must be taken of the mode of onset of the convulsion, and the site of the initial spasm carefully noted. If the sensory symptoms preceding the convulsion are very definite, the irritant focus may prove to be in the anterior parietal cortex, whereas initial motor spasm will indicate a lesion in the particular limb area in the ascending frontal convolution. After trephining and removal of a considerable bone-flap, careful unipolar faradic exploration of the cortex with a weak current must be undertaken, under ether anæsthesia, in order to reproduce the motor symptoms observable at the commencement of the convulsions, and excision of this area to the depth of about ¼ inch may be followed by cessation of the convulsions and great improvement in the condition of the patient. Considerable difference of opinion exists as to the benefit of such operations, some observers declaring that any such operation must leave an irritant scar behind which will reproduce the symptoms. Such, however, is not the experience of certain operating surgeons, who declare that bad results are due to the irritant focus not having been carefully located by faradic stimulation of the diseased focus.

Contrecoup

A phenomenon which is not infrequently seen in the severer cases of contusion and laceration of the brain is that known as **contrecoup,** in which a portion of the cortex on the opposite side of the brain to the direct injury is bruised or lacerated. This appears to be the result of transmission of the shock of the convulsions through the brain substance itself, by which means the opposite side of the brain is damaged against the containing bony wall. Such an indirect lesion may accompany laceration and contusion of the brain directly below the site of the blow, and in some cases the lesion due to contrecoup may appear to be the more important. This is particularly likely to happen when the right side of the head is struck, the lesion of contrecoup in those cases not infrequently damaging the cortical centres for speech on the left side of the brain, in either the frontal, temporo-sphenoidal, or lower parietal regions. If the contusion or laceration due to direct violence should affect the right temporal, parietal, or anterior frontal lobes, it is quite possible that no physical signs may be present to indicate such lesion of the nervous system, while a lesion due to contrecoup may perhaps involve either of the speech centres on the left side of the brain, as above stated, or parts of the left motor cortex itself. In such a case the physical signs of damage of the nervous system due to the contrecoup will be very obvious. The treatment in such cases will be the same as in the cases of direct convulsions, and no operative interference is to be permitted in the earlier stages, unless definite signs of compression of the brain are present.

Treatment of Cerebral Injuries

In the earlier treatment of cerebral injuries producing concussion, fractured base, contusion, laceration, and cerebral hæmorrhage, the most important point is to avoid doing too much, and to leave the patient alone in perfect rest as far as possible, and never to operate unless such interference is urgently called for by unmistakable symptoms. Many a life has been lost, or irreparable damage done, by too active surgical interference in cases where coma is prolonged, or especially if Jacksonian convulsions develop. It is often better to leave a patient lying where he has fallen, without disturbing him, until a proper ambulance and the necessary means for moving him have been secured, rather than to drag him into a neighbouring house or a chemist's shop, where the air is warmer and more likely to promote increased rapidity of the circulation, and therefore the liability to increase the cerebral hæmorrhage or œdema. The chill of exposure is far more likely to do good than harm for the first few quarters of an hour, by its effect in diminishing œdema and hæmorrhage. No attempt should be made to administer restoratives in the shape of food or brandy poured into the mouth, for at least six hours after the accident, nor should vasomotor stimulants such as ether, brandy, or pituitary extract be administered hypodermically, unless a flickering pulse and failing respiration indicate imminent danger of failure of the vital centres.

The clothes must be cut off, and the patient carefully put to bed with as little disturbance as possible, the head being supported on a pillow, so as to raise it very slightly above the level of the chest. The scalp must be carefully searched for wounds, which must be gently sponged out with some mild antiseptic, such as boracic lotion, and painted with two per cent iodine in spirit. Suture of such scalp wounds is best delayed for several hours, after securing any bleeding-points, in order to minimize shock, and even then should not be done without infiltrating the area of the wound with 0·3 per cent novocain solution. When searching the scalp for injury, punctured wounds and depressed fracture must be carefully looked for, as these are probably urgent and will require immediate surgical attention by trephining. Such an operation, again, should be done after infiltration of the whole area to be dealt with by novocain solution, as the danger of shock is thereby considerably lessened. Ether or chloroform anæsthesia will usually not be required, as the patient is already semi- or wholly comatose, and trephining under novocain infiltration is not a painful operation, and can often be done in an ordinary patient without any general anæsthetic.

Punctured wounds are likely to be especially dangerous for several reasons. Septic material may be carried through from the scalp into the pia-arachnoid and cerebral substance, or the inner table of the skull, which is so much more brittle than the outer portion, may be splintered, and portions forced into the outer cortex, causing irritant symptoms, or possibly meningitis or later cerebral abscess. Lumbar puncture will be of service in demonstrating the onset of meningitis. This serious complication will be indicated by a turbid cerebro-spinal fluid, containing polynuclear leucocytes and micro-organisms, usually streptococci or staphylococci. As soon as the scalp has been cleaned up and a light dressing bandaged on, an ice-bag may be applied over the dressing, though it must be hung so as not to press by its weight upon the head. The bedclothes should be not too heavy, and hot bottles, well wrapped in flannel, should be placed one on each side of the feet and legs.

Care must be taken that the bottles are not too hot, or serious burns may be produced while the patient is unconscious. If there has been much loss of blood, and if the pulse is weak and irregular, a saline enema of 1½ pints normal saline, containing 4 oz. of glucose dissolved in it, should be administered, and later, attempts must be made by spoon-feeding with the usual liquid nourishment—milk, soups, eggs and milk, &c. Attention must be paid to the sphincters, as retention of urine is likely to occur, and regular catheterization may be required twice a day; usually, however, reflex incontinence of both sphincters is set up, and careful nursing will be necessary to keep the patient clean and dry. Indeed in cases of prolonged unconsciousness from cerebral injury, care of the skin of the back and the keeping of the patient clean, with the avoidance of bedsores, is an important item of the treatment. If the patient is a man, the use of a urine bottle will generally keep him dry, though in the case of a woman it is quite impossible to do so.

Restlessness and delirium, with accompanying insomnia, are difficult to combat, as morphia is not without danger in coma. Bromide and hyoscine, or sulphonal, should first be tried, but chloral should be avoided for regular use. Insomnia, however, must be overcome, and opium in 15-minim doses of the tincture may be necessary, or the patient's strength will be worn out for want of sleep.

After-treatment

The after-management of cases of concussion and cerebral injury is scarcely less important than the immediate diagnosis and treatment. Rest in bed in a darkened room for at least a month should be insisted on, when the patient has been unconscious for an hour or more, and avoidance of all mental fatigue, including visitors and talking, must be carefully guarded against. The question to be decided, as to when a given patient may return to his ordinary mode of life and work, must be judged on its merits in each case. Broadly speaking, if the daily work necessitates keen and prolonged attention, six months' holiday will be advisable, and a change of scene, with quiet outdoor employment, such as fishing, boating, golfing, and later perhaps travel abroad, may best fit the brain for a later resumption of full work. In many cases, however, six months is not enough, and a year or two may elapse before complete recovery is attained.

Remote Effects

Persistent headache is one of the commonest symptoms to be complained of after concussion and contusion of the brain. This may be due to a chronic meningitis resulting from fracture of the skull setting up irritation and inflammatory exudation of the meninges, or hæmorrhage on the surface may not be fully absorbed, leaving behind an organized clot adherent both to the dura and pia-arachnoid. In older persons, moreover, persistent headache following an injury to the head may be largely due to arteriosclerosis, though headache may not have been previously a prominent symptom. More important, perhaps, to recognize will be a gummatous endarteritis, periarteritis, and meningitis, which may follow directly after a severe blow upon the head in a syphilitic subject, though the disease may previously have been latent. The recognition of this cause may be suggested by the nocturnal intensity of the headache, by lymphocytosis noticeable in the cerebro-spinal fluid, and by a positive Wassermann reaction in the blood, and perhaps in the fluid also. A late appearance of diplopia and other symptoms of progressive lesion of the cranial nerves will also be strongly suggestive of a syphilitic cause. Should this diagnosis be established, an immediate intravenous injection of salvarsan, or of a British equivalent, Kharsivan, which I have found equally efficacious, should be given, and repeated in three weeks' time if necessary, employing the interval in administering biniodide of mercury by the mouth, or mercurial inunction.

In a certain number of cases cerebral tumour dates the development of its symptoms from shortly after an injury, and this has occurred with sufficient frequency to associate a blow upon the head as one of the causes of tumour of the brain. I have seen such cerebral tumour, a gliosarcoma, following an injury, infiltrate the skull over a considerable area beneath the site of the original blow, in addition to destroying a large area of the cortex and brain substance beneath. Simple exostoses and bony hyperplasia also may result from fracture of the brittle inner table, without any fracture of the external bony wall. Persistent headache may result from this cause, and even slow compression of the cortex in rare cases may ensue with its dependent paralytic phenomena.

A skiagram of the skull should always be taken in cases of severe head injury.

Neurasthenic headache, usually vertical, is another very common result of head injuries (see p. 99).

FACIAL NEURALGIA

A rare sequel to severe blows upon the head is chronic neuralgia, which may take the form of continuous lancinating pain in the face or forehead, or it may be paroxysmal, of the tic douloureux type. Fractured base, as stated above (p. 11), may damage the Gasserian ganglion and the fifth nerve, and more or less persistent facial neuralgia, with recurrent exacerbations of pain, will then be referred to the anæsthetic area supplied by the trigeminal nerve. In numerous cases I have seen severe blows on the jaw or side of the head followed after a short interval, varying from two hours to two weeks, by true paroxysmal trigeminal neuralgia, or tic douloureux. In this type there is no loss of sensation, and between the bouts of pain the patient may be perfectly comfortable. Again, falls upon the face may produce local neuritis of a branch of the fifth nerve, such as the supra-orbital or infra-orbital, instances of both of which I have met with, with numbness and diminished sensibility of the area supplied by the nerve, and paræsthesiæ referred to the part, such as crawling and tickling sensations, but usually no special pain.

Cerebral abscess is an infrequent sequel to fractured base, and I have known death from this cause produced a year after the injury, the patient having suffered from recurrent headaches, but not from persistent pain, after he had recovered from the immediate effects of the blow.

In this case an abscess was found in the frontal lobe, and was probably the result of infection from the upper nasal cavity, the fracture having passed through the cribriform plate. More common is abscess in the temporal lobe, or in one lateral lobe of the cerebellum, the infection in this case being derived usually from the middle ear. On account of this danger, and still more to avoid the danger of a septic meningitis being set up, the external auditory meatus should never be syringed out in cases of cerebral injury, even if full of blood-clot, but should instead be mopped out carefully with pledgets of cotton-wool soaked in boracic lotion.

Cerebral abscess from middle-ear infection may be found post mortem without any direct tract of infection or intervening meningitis being visible, and its recognition will depend on the usual manifestations of intracranial pressure, combined with the localizing signs due to disease of the parts. Optic neuritis may or may not be present. Temporal abscess may cause no distinctive phenomena, unless pressing deeply inwards upon the internal capsule, when hemiplegia will be likely to develop slowly. If the left temporal lobe be thus involved, symptoms of sensory aphasia, word-deafness or word-blindness, may be found.

The treatment of cerebral abscess is straightforward, once the diagnosis is established, operation and draining of the cavity being the only remedy offering a chance of life.

Cerebral thrombosis, either of arteries or of veins and sinuses, is another occasional late result of severe injury to the head, the symptoms varying according to the area of brain supplied by the damaged vessel. The symptoms will usually develop more or less gradually in from one to several hours, though occasionally the onset may be sudden. When extensive thrombosis of the sinuses takes place the patient may be deeply comatose, and retinal hæmorrhages, with perhaps proptosis on each side, may be present, owing to obstruction of the cavernous sinus, and recurrent convulsions may ensue, death from this cause being not unusual.

CHAPTER II

INJURIES OF THE SPINAL CORD

Spinal Concussion.—Owing to the more secure protection of the spinal cord by its method of suspension in a water-bed, enclosed in a semi-rigid canal composed of the bodies of the vertebræ and intervertebral disks in front, and of the laminæ and neural arches, gross injury and contusion of the spinal cord from concussion is comparatively rare, though symptoms of temporary paraplegia of varying degrees of severity may follow a severe fall or blow upon the back. Jarring of the vertebral column also, as by a heavy fall upon the feet, may cause a temporary derangement of the lumbar cord, with weakness and paræsthesiæ of the legs, though it is often hard to draw the line between symptoms due to direct injury inhibiting the spinal functions, and those of hysterical origin. Sphincter incontinence and extensor plantar reflex may be taken as decisive of an organic lesion, even though such a lesion amounts only to temporary nutritional disturbance. As a result of more severe blows actual hæmorrhage may take place, either into the substance of the spinal cord or into the membranes, or again the hæmorrhage may be extra-dural. Intraspinal hæmorrhage will produce instant paraplegia, both motor and sensory, in proportion to the severity of the extravasation.

Not infrequently there may be more than one point of hæmorrhage. If the bleeding is located in the central grey matter, it may travel vertically for a space of two or more segments at the base of the posterior horn, producing weakness of the lower extremities by pressure laterally upon the pyramidal tracts, with increased knee-jerks, ankle clonus, and extensor plantar reflexes, with a dissociated anæsthesia, tactile sensation being preserved, but with considerable loss of sensation to pain and to heat and cold over the lower extremities, and of the trunk up to the level of the damaged segment. In such a case motor power may be considerably recovered as the hæmorrhage is absorbed, though the loss to pin-prick and to sensations of warmth and cold may be permanently diminished. If the hæmorrhage involves the anterior horns of the grey matter, then localized muscular paralysis with subsequent atrophy will be superadded to the foregoing clinical signs. This localized muscular paralysis will not be obvious, however, unless the lesion involves either the cervical or lumbo-sacral enlargement, in which cases wasting of muscular groups in the upper or lower extremity will be very noticeable. The grouping of the paralysed muscles will correspond to that of spinal segments, such as the deltoid, spinati, biceps, and supinator longus in lesions of the fifth cervical level, or the triceps, serratus magnus, lower pectoral, and latissimus dorsi when the sixth cervical segment is involved.

A not uncommon injury of this nature is hæmorrhage into the sacral cord at the second and third sacral levels, resulting from a heavy fall in a sitting posture from a height of a few feet. In these cases there is paralysis and wasting of the gastrocnemius and of the glutei and anal sphincters, with resulting retention and incontinence of urine, and inability to raise the body on the toes, thus causing a heavy flat-footed gait. The area of the anæsthesia will involve the nates and the genitals, perineum, and a strip down the back of the thigh and calf on each side, corresponding to the second sacral distribution, while the muscular wasting will involve the buttocks and calves, and the anal sphincter will be patulous. The chief difficulties these patients have to contend with is the management of the sphincters, as permanent use of the catheter may be necessary, the constipation requiring enemata. Perhaps the most satisfactory way of dealing with the bowel trouble is to give an enema in the evening, in order to avoid a recurrent slight action occurring during the daytime. Additional help will be the pressure of a pad of wool over the anus, supported by a sanitary towel attached to a belt.

Intrameningeal Spinal Hæmorrhage

Hæmorrhage from a superficial vessel in the pia-arachnoid may occur as the result either of a blow upon the back, from the jar of falling from a height, or from the strain of a heavy muscular effort. The rupture generally takes place from a vein, and in the last cause mentioned of muscular effort the venous blood-pressure is suddenly and considerably raised by the breath being held at the same time as the forced expiratory effort. Such intrameningeal hæmorrhage may be either intracranial or intraspinal, sudden pain in the back of the head being the initial symptom if the bleeding is intracranial, though the pain is referred further down the spine according as the hæmorrhage is intraspinal. The pain may be excruciating, rendering the patient almost helpless, and will persist for many hours, gradually diminishing locally, though radiating pains will a day or two later appear in the extremities, almost invariably the lower limbs, as the blood is carried downward by the cerebro-spinal fluid, and may coagulate, forming a thick clot around the roots of the cauda equina. In the latter event, rigidity of the lower spine and pain on movement will last for many weeks and even years, unless the clot be removed surgically.

The diagnosis will rest in such a case upon the history of absolutely sudden onset of violent pain, followed later by radiating pains in the lower extremities, and signs of irritation of the posterior roots, as shown by the rigidity of the lower spine, and zones of hyperæsthesia and possible analgesia along some of the lumbo-sacral root areas, with diminution of the knee or Achilles jerks. Lumbar puncture will confirm the diagnosis, for within the first three days cerebro-spinal fluid intimately mixed with blood may be withdrawn, though after the first few days free blood is no longer found, but the cerebro-spinal fluid will be tinted yellow from the alteration of the blood pigment, which will now react to the chemical test for bilirubin. Though intrameningeal hæmorrhage may thus form a clot surrounding the cauda equina, it does not give rise to symptoms of direct pressure upon the cord. This result may, however, happen when the hæmorrhage is extra-dural, and above the second lumbar vertebra, when the bulging inward of the theca by the blood-clot on its outer surface may press upon the spinal cord, producing paraplegia. The localization of the level of this lesion should be comparatively easy, owing to the level of the upper limit of the anæsthesia produced, indicating the spinal segment which is compressed.

Thus anæthesia reaching up to the level of the navel only will indicate a lesion at the level of the tenth dorsal segment. In such a case there will be anæsthesia of the whole of both lower extremities and of the lower part of the trunk up to the level of the navel, the lower extremities will be paralysed, the knee-jerks and Achilles jerks increased, with extensor plantar reflex, while the sphincters will no longer be under voluntary control. If these symptoms persist without improvement for 24 hours, laminectomy should be performed at the level of the spinal column corresponding to the segment of the spinal cord compressed. Roughly, when the anæsthesia reaches to the navel the lesion in the spinal cord will lie from 3½ to 4 inches above the upper limit of the zone of anæsthesia on the back.

Fractures and Fracture-dislocations of the Spinal Column

Far graver in its effects than hæmorrhage into the meninges are the lesions produced in the spinal cord by fractures through the bodies of the vertebræ, or through the laminæ, or by fracture-dislocations in which not only one vertebral body may be crushed but the neighbouring vertebra, usually the lower, is pushed violently backwards against the spinal cord, pressing on or crushing it completely. Rarely a fracture may be vertical through the middle of the body, in which case there is no dislocation or deformity of the parts. Yet I have seen severe and fatal hæmorrhage within the spinal cord produced as a result of this injury in the sixth cervical vertebra.

More usually the fracture is more or less transverse, and the crushing of the anterior portion of the body will produce an angular curvature visible and palpable from behind. Rarely the fracture may involve the articular processes and the laminæ, with little or no damage of the vertebral bodies, and in such a case the cord may be compressed from behind by a depressed portion of the fractured lamina. The resulting paraplegia in such a case is much more likely to be relieved or even cured completely by an operation for laminectomy and removal of the depressed bone, than in those cases where the cord has been damaged by the backward thrust of a vertebral body. Unfortunately the favourable cases in which laminectomy is able to remove the cause of compression, and to cure the consequent paraplegia, are only too rare, though occasionally a brilliant result is achieved. More often than not the spinal cord has been irretrievably damaged by the sudden violent compression, and by consequent hæmorrhages which have taken place within it.

Broadly speaking, the severity of the symptoms varies with the height of the lesion in the spinal cord, lesions of the lower dorsal and lumbar regions being much less dangerous to life than injuries causing destruction of the cord in the upper dorsal, and particularly in the cervical regions. Fractures above the level of the sixth cervical vertebra will cause almost immediate death from paralysis of the respiration, as the phrenic nerve is mainly supplied from the fourth cervical segment, which lies opposite the body of the fifth cervical vertebra. Consequently, if fracture occurred through the fifth cervical vertebra, the fourth cervical segment of the cord is liable to injury, and consequently paralysis of the diaphragm will follow, in addition to the paralysis of the intercostals which are supplied from lower down the cord. With lesions of the cervical cord, in addition to paralysis of the lower extremities, trunk, and intercostal muscles, there will be paralysis of certain groups of muscles in the upper extremity and wasting of other muscles of the upper limb and shoulder-girdle according to the segment affected. Symptoms specially significant of cervical lesions are pyrexia and obstinate priapism. If the transverse lesion is complete the pupils will be medium small, and will not dilate well when shaded, though there will be slight though brisk reaction to light. The upper lids also are slightly drooped, due to partial ptosis, these ocular symptoms being due to paralysis of the sympathetic fibres which are running down the spinal cord towards their exit by the first dorsal anterior roots to join the cervical sympathetic nerve. In transverse lesions of the fifth cervical segment, as may be produced by a fracture of the sixth cervical vertebra, the upper extremities will lie adducted to the side and rotated inwards, with the forearms extended. This position is due to the local destruction in the fifth cervical segment of the anterior horn nuclei supplying the deltoid, spinati, biceps, and supinator group of muscles.

If the patient lives sufficiently long these muscles will atrophy, showing reaction of degeneration to electrical testing, while the remaining muscles of the upper extremity, such as the triceps and forearm muscles, will develop rigidity, with increased reflexes. Transverse lesions through the next segment below, the sixth cervical level of the cord, will cause a different muscular paralysis in the upper extremity, the triceps, latissimus, lower pectoral, and serratus magnus suffering from atrophic paralysis. The limb will assume a characteristic position known as Thorburn's position, as the deltoid and biceps by their unopposed tonic action will abduct the arm from the side and flex the forearm acutely.

The segmental muscular supply of the cord above indicated must not be taken to be absolutely precise, as each muscle no doubt receives fibres from more than one segment, yet the above clinical picture is fairly accurate. Speaking broadly, in the same way the extensor muscles of the fingers may be looked upon as represented in the seventh cervical segment of the cord, though the radial extensors of the wrist are undoubtedly represented higher, often in the fifth cervical with the deltoid and biceps.

The flexors of the fingers and the ulnar flexor of the wrist roughly represent the eighth cervical segment, though the flexor carpi radialis probably belongs to the sixth and seventh segments. The intrinsic musculature of the hand corresponds to the first dorsal segment. Just as atrophic paralysis and reaction of degeneration of one or other of these groups of muscles will indicate the level of the lesion of the cervical cord, so will the anæsthesia resulting from the lesion confirm the diagnosis. Thus, anæsthesia of the lower extremities and trunk, reaching up to the second rib and including the inner side of the arm and forearm down to the level of the wrist, but not invading the hand itself, will indicate the first dorsal segment as the highest segment damaged. If the eighth cervical segment is damaged, the anæsthesia will invade the inner side of the hand and little and ring fingers, corresponding to the ulnar nerve distribution. The seventh cervical segment probably supplies the index and middle fingers and the outer half of the ring finger, whereas the thumb is supplied by the sixth cervical segment. If the lesion is through the sixth cervical segment, and is fairly complete and transverse, the upper limbs will assume Thorburn's position as above described, and the whole of the hand and most of the forearm and arm will be anæsthetic, with the exception of a strip on the radial side running down as far as the junction of the wrist and ball of the thumb. Over this area the patient will be able to feel light touches and pin-pricks, and this is no doubt supplied in part by the fifth cervical segment. It should be remembered, however, that considerable overlapping takes place in all these root supply areas, and any given zone is probably supplied by at least three segments, if not by more. The sensory supply of the fifth cervical segment is a less important function of this root than is its motor supply, and so well supplied is this area of skin along the radial side of the arm and forearm by the lower roots, the sixth and seventh cervical, that in cases of injury of the fifth cervical root alone outside the cord, the resulting symptoms are those of purely motor paralysis of the deltoid and biceps group, no trace of any anæsthesia being detectable in such cases. Transverse lesions destroying the fifth cervical segment cause complete anæsthesia of the upper extremities up to the points of the shoulders. Anæsthesia reaching up to just below the nipples will indicate a lesion through the fifth dorsal segment, the xiphisternum corresponding to the seventh dorsal, the navel to the junction of the ninth and tenth dorsals, whereas on the lower extremity, roughly speaking, the lumbar roots run down, in order, the front of the thigh and the leg, and the sacral roots supply the sole of the foot and the back of the leg and the thigh, the nates and perineum. Thus, the middle front of the thigh corresponds to the third lumbar segment, the sides of the thigh and the inside of the leg and foot to the fourth lumbar, while the fifth lumbar supplies the front and outside of the leg and the dorsum of the foot and ankle. The first sacral supplies the sole of the foot, the upper surface of the toes, and the outside of the foot and heel, roughly the 'golosh' area; the second sacral corresponds almost to the small sciatic nerve, a strip of skin along the back of the calf and thigh up to the buttock. The third sacral supplies the nates and genitals.

Of the deep reflexes of the lower extremities, the knee-jerks correspond to the third and fourth lumbar segments, and the Achilles jerks to the second sacral. Of the skin reflexes, the cremaster reflex is first lumbar and the plantar reflex is first sacral, while the sphincter control is mainly represented in the third sacral segment.

Focal and unilateral lesions of the spinal cord are not uncommon as the result of direct violence from fracture of the vertebral bodies or laminæ, and similar damage to the cord not infrequently results from penetrating wounds, such as from gunshot injury or by stabbing. Partial transverse lesions may involve either one-half of the cross-section of the spinal cord, in which case we shall obtain a clinical picture of a truly unilateral lesion, or, as is more usually the case, the injury extends across the middle line to involve one or more tracts upon the other side also, such as the posterior columns. The symptoms due to a unilateral lesion are sometimes known as Brown-Séquard paralysis, after the neurologist who described them many years ago. There will be motor paralysis upon the same side as the lesion, namely, atrophic paralysis of the group of muscles represented in the particular half-segment destroyed, together with spastic paralysis of the lower extremities on the same side. This is due to the crossed pyramidal tract being injured by the lesion, the motor fibres having already decussated higher up in the medulla. The knee and Achilles jerks will therefore be increased on the paralysed side, with ankle clonus, extensor plantar reflex, and diminution or loss of the abdominal skin reflex. Sensation to touch and pin-prick will not be affected upon the lower limb of the same side ; there will be loss of sensation of position of the joints and toes, and also loss of perception to vibration, while there will be loss to pain and to temperature sense upon the lower extremity of the opposite side, and upon the opposite side of the trunk up to within a varying distance from the level of the lesion. Tactile sensibility may be little affected, or not at all, on either side, and the patient for that reason may not be aware of any loss of feeling until tested. There is, however, what is called crossed analgesia and therm-anæsthesia, involving the unparalysed limb on the other side. This loss of pain and temperature sense on the opposite side is due to the fact that the nerve fibres carrying pain and heat sensations, soon after their entry into the cord by the posterior roots, cross over and run up the other side of the cord in the ascending antero-lateral tract. Sometimes this analgesia reaches up to and will localise the actual segment destroyed, though in other cases it ceases at a level several segments below. In unilateral lesions the sphincters are, as a rule, comparatively slightly affected. When the lesion is not strictly unilateral, but has damaged the posterior columns of both sides, then there will be marked tactile anæsthesia on both sides below the level of the lesion, and the difference in sensibility to pain and heat on the two lower extremities may not be very pronounced.

Occasionally a **focal** injury from hæmorrhage may occur, damaging one particular area of grey or white matter. In this way, if the lesion is limited to the anterior horn of one side, atrophic paralysis of certain muscles will ensue, while if the central grey matter is damaged alone, the result may be analgesia and thermanæsthesia of one or both lower extremities, owing to the nerve fibres carrying heat and pain sensations being injured where they are crossing to the opposite side.

It will of course be obvious that atrophic paralysis of muscles representing a definite spinal group is easily recognizable only if the lesion occurs either in the cervical or lumbo-sacral enlargements. When the lesion occurs in a dorsal segment it will be difficult or impossible to find on examination atrophy of portions of the trunk muscles.

Total transverse lesions produce a different clinical picture to that resulting from partial incomplete transverse lesions of the cord. As a result of the total transverse lesion, there will necessarily be absolute and complete motor and sensory paralysis below the level of the lesion, the patient being totally unable to perceive any sensory stimuli applied to the parts below, with the exception of jars on the bones which may be transmitted vertically along the spinal column. In these cases the paralysis of the lower extremities is flaccid, there being no appearance of any muscular rigidity, and the knee and Achilles jerks remain permanently absent. The plantar reflex is often completely absent in total transverse lesions, though in a certain number of these cases a slight though distinct plantar reflex can be obtained, which may be either flexor or extensor, even within an hour or two after the occurrence of the injury. Even the abdominal and cremasteric reflexes may be preserved in some cases. The reflex effect upon the vital centres of total transverse lesions, whether the result of fracture of the spine or from bullet wound, is usually so severe that death results within a few hours or days. In a few cases, however, this may be postponed for months, in which case extreme muscular atrophy of the lower extremities takes place, the muscles no longer reacting either to faradism or to the galvanic current. The management of these cases is chiefly a matter of careful nursing, the difficulty being to avoid the occurrence of bedsores from pressure upon the bony prominences, such as the sacrum, buttocks, the great trochanters, and the heels. The incontinence of the sphincters, and the total inability of the patient to give warning when help is required for these matters, greatly increases the difficulty of keeping the patient clean and dry, and the skin in good condition. A water or air mattress is essential to equalize pressure, and rubbing the skin twice daily with spirit, and then with an antiseptic dusting-powder of boracic acid and starch, is of great service. Rubber ring cushions and other devices must be tried to avoid undue pressure upon the heels, sacrum, and other parts. Sometimes bedsores occur in spite of the greatest care, and the skin having lost apparently its vitality, attempts at repair are slight, and the bedsore may progress deeply, involving the bone underneath. Pyrexia in these cases will indicate either cystitis or collections of pus in sinuses communicating with the bedsore, and those may be dealt with *secundum artem.* When the trophic ulceration is involving the bone of the sacrum, there is great danger of infection spreading through to the spinal meninges and setting up an infective meningitis. When this stage has been reached death is a matter of a day or two only.

Partial transverse lesions are less dangerous to life in their immediate effects, though the intense rigidity of the lower extremities, with flexor and adductor spasms of the knees and hips, makes the nursing very difficult, and is a source of considerable discomfort to the patients themselves, the spasms being often rather sudden and violent, keeping them awake or waking them up at night. There is somewhat less risk of bedsores according as the cord lesion is less severe, but the chief danger in the after-treatment is the management of the bladder, owing to the danger of cystitis being developed. So long as reflex incontinence is present, and the bladder is well emptied, a catheter should never be passed ; but if the bladder remains overfull, and dribbling incontinence from overflow is set up, then catheterization every twelve hours is necessary, until the reflex tone of the bladder is established, if at all. With even the greatest care in asepsis, it is almost impossible to avoid bacterial infection of the bladder in such cases, and treatment by hexamine and perhaps irrigation and drainage will be necessary. With bacillus coli infection the urine may still remain acid, though full of mucus and pus. If there is much intermittent pyrexia, in spite of treating the bladder as described, the probabilities are that the infection has ascended the ureters, and that a septic pyelitis has developed. This is a very serious danger, leading to pyonephrosis, uræmia, and death. Indeed, in chronic nervous diseases, in which the posterior columns of the spinal cord are affected, the chief danger to life lies through infection of the bladder, and consequent surgical kidney and uræmia.

Remote Effects of Spinal Injury

It has for long been considered doubtful, and even now some physicians will not concede the point, whether definite system diseases of the spinal cord, such as progressive muscular atrophy, tabes dorsalis, &c., can be held to be due to injuries or blows upon the back. Personally I have no doubt at all upon this point, and it is becoming generally accepted now that such may be the case. In the ætiology of tabes dorsalis, it may be taken as axiomatic that previous syphilitic infection, generally acquired, but sometimes congenital, is necessary for the development of the disease. Formerly looked upon as parasyphilitic, or a late post-syphilitic degeneration of the nervous elements, not dependent upon any syphilitic gummatous lesion, and therefore not benefited by ordinary anti-syphilitic treatment by mercury and by iodide, this disease has of late years come to be regarded as truly syphilitic, that is to say the result of actual lesions of the meninges and blood-vessels dependent upon the syphilitic virus, in which the spirochæte may be found. Although this micro-organism is very difficult to demonstrate in tabetic lesions, yet in general paralysis, a cerebral disease which is closely allied to tabes dorsalis, and not infrequently is combined with it, when it is known as tabo-paralysis, the Spirochæte pallida of Schaudinn is almost constantly to be found in the cerebral substance of the frontal lobes, thus proving the disease to be a truly syphilitic process, dependent upon the actual virus, and not a late post-syphilitic toxic degeneration of the nervous system. With this newer view of tabes and general paralysis it is not hard to reconcile the statement that either of these diseases may develop as a sequel to an injury. For many years it has been a commonplace of surgical knowledge that gummata and other progressive syphilitic lesions are liable to follow direct injury to tissue, in an individual who has previously suffered from syphilis, but in whom at the time the disease was completely latent, and perhaps thought to have been cured. Though intensive anti-syphilitic treatment on modern methods has produced most hopeful results in the treatment of tabes dorsalis, yet so far no such good result can be claimed in the treatment of general paralysis. Possibly the cause for this depends upon the deeply lying position of the spirochætes in the brain substance, and the difficulty, if not impossibility, of bringing bactericidal agents to bear upon them.

Another disease which in many cases has been ascribed to direct injury, such as a blow upon the back, or direct injury to a limb, is progressive muscular atrophy, a chronic degeneration of the anterior horn cells, usually starting in the cervical enlargement. The dependence of this degeneration upon indirect violence is difficult to substantiate, yet such a history has been obtained sufficiently frequently to make the association of the disease with injury more than a mere coincidence. Combined sclerosis, spinal gummata, and other tumours have also followed injuries of the spine, and though the evidence connecting the development of gummata with antecedent injury may be considered as conclusive, it is an open question how far an injury may be believed to be the cause of spinal tumours or combined sclerosis. The decision must be carefully weighed in each case, and will depend partly upon the severity of the injury, the previous nervous history of the patient and his family, and the interval of time separating the injury from the development of the first symptoms of the disease.

Pain in the back is one of the commonest persistent symptoms following spinal injury, and is often extremely difficult to treat. The pain is commonly situate in the lumbar region, usually to one side or the other near the middle line, where there is generally tenderness over one or more points. In many cases the injury, such as a slight fall, has been totally insufficient to have caused any gross injury of the spinal cord or of its membranes, and the diagnosis will usually rest between an hysterical pain and a ruptured slip of muscle with a persistent fibrositis. In some cases the injury gives rise to osteo-arthritis, and I have seen persistent pain and rigidity of the neck due to this cause in a patient who had been thrown out of her motor on her back in the road. Skiagrams showed complete synostosis of the third and fourth cervical vertebræ. Osteo-arthritis of the larger joints is well known to occur as a fairly frequent result of injury, and in all probability this disease occurs as the result of falls upon the back more frequently than has been hitherto thought. Excruciating root pains will result from narrowing of the intervertebral foramina due to arthritis ; the clue to this diagnosis will be found in the rigidity of the spine, and skiagrams may show little or no lipping of the bone until the spondylitis is advanced. The treatment of these cases is extremely difficult, most benefit probably being obtained by courses of hot saline baths and sulphur medication.

In cases of chronic pain in the back following injury, where the mobility of the spine may be taken to exclude osteo-arthritis, the pain is usually hysterical, or due to chronic fibrositis, the latter often of a rheumatic type. The diagnosis of hysteria as the cause in such cases should be made with caution, and at best is merely tentative. If other hysterical stigmata such as hemianæsthesia, or hysterical motor paralysis, aphonia, and the like be present at the same time, the diagnosis of hysteria as the cause of the pain in the back will be more than justified, though even then the pain may have quite a different origin. Counter-irritation of the painful spot will in either case be a useful treatment, whether it is due to hysteria or to a rheumatic fibrositis, and for this purpose the actual cautery applied over the tender spot will often work like a charm. In other cases where the diagnosis of hysteria need not be considered, and where intense local tenderness is a constant feature, good results may often be obtained by local injection of the tender point with a two per cent novocain solution, followed by strong alcohol. In doing this a fine needle of about 2½ inches in length should be chosen, and after anæsthetizing the skin by puncture with a fine hypodermic needle and novocain, the larger needle is pushed in through the same puncture made by the hypodermic needle, and pressed down until it reaches the bone underneath, such as the transverse process or the vertebral lamina, or the iliac bone, and novocain then injected and followed by 7 minims of 90 per cent alcohol. Ionization with salicylate of soda and with iodine has often been tried for these cases, usually without much benefit, and better results are likely to be obtained with combined faradism and galvanism, or with faradism alone as strong as it can be borne. Many such cases of chronic pain in the back following slight injuries, where no evidence of organic lesion can be found, and in which there is neither albuminuria nor glycosuria which might give rise to chronic neuritis, occur in compensation cases where an action for damages is brought on account of an injury. The majority of these cases occur under the Workmen's Compensation Act, and undoubtedly malingering is accountable for a certain number of them. Genuine hysteria will account for others.

It is often extremely difficult to separate malingering from hysteria, and indeed many practitioners refuse to consider hysteria as a genuine neurosis apart from an intention to deceive. The presence of hysterical stigmata will aid the diagnosis, namely, the presence of patches of anæsthesia, hemianæsthesia, paraplegia with normal deep reflexes, absence of the plantar reflexes with brisk abdominal reflexes, aphonia, contraction of visual fields, vertical headache, persistent tremor, &c. Undoubtedly the mental worry of legal negotiations accentuates and keeps up hysterical phenomena, and they often disappear at once after the conclusion of compensation proceedings. One point of considerable value in the differentiation of hysterical phenomena from organic lesions due to direct injury, is the fact that, in the majority of cases, hysterical phenomena are not developed at once after the injury, but appear from a few days to several weeks afterwards.

Lesions of Cauda Equina.—Below the termination of the sacral cord at the lower border of the first lumbar vertebra, the anterior and posterior roots from the second lumbar downwards descend to their respective foramina of exit, forming a bunch of nerves known as the cauda equina, the lowest sacral roots being innermost. Fractures of the spine or bullet wounds through the lower lumbar or sacral region will be liable to cause flaccid paraplegia, with motor and sensory paralysis and muscular wasting, corresponding to the damaged roots. There will be loss of the deep and superficial reflexes, and incontinence of urine and fæces, with a patulous sphincter ani. If the injury is below the level of the fourth lumbar vertebra, the knee-jerks will be unaffected, though the Achilles jerks will be lost. Sacral injuries will cause ‘ saddle anæsthesia ’, and generally the condition described on p. 33. A lesion of the lumbar spinal cord will cause loss of knee-jerk, though ankle clonus may be present, and extensor plantar reflex. Moreover, the external sphincter ani will contract smartly if pricked with a pin, though it may be totally anæsthetic. Operation may be undertaken in cauda equina lesions to remove fragments of bone or other foreign body causing compression, though it is useless to suture divided nerve roots.

CHAPTER III

BRACHIAL PLEXUS INJURIES

SEVERE paralysis of the upper extremity not infrequently results from injury to the spinal nerves after their exit from the intervertebral foramina, before they weave together into that meshwork of nerves known as the brachial plexus, and from which emerge the main nerve trunks to the shoulder and arm. The spinal nerves which form the brachial plexus are five in number, namely, the ventral branches of the fifth, sixth, seventh, and eighth cervical and the first dorsal, with small branches also from the fourth cervical and the second dorsal. It is the uppermost and the lowest of these nerves which are most exposed to damage, owing to their more sloping direction, injuries of the seventh and eighth cervicals being far less common except when the violence is extreme, as may occur for example from the wrenching of a limb which has been caught in revolving machinery. One well-known type of paralysis is that usually known as Erb's palsy, in which the fifth cervical nerve particularly, and to a less extent the sixth cervical, are violently stretched, or even ruptured, as the result of falls upon the point of the shoulder. In such a case, when the arm is not put out by the patient to save himself, but remains by the side, the shoulder is suddenly and violently depressed by the force of the blow, and the head is pulled to the opposite side, thus putting sudden violent tension upon the uppermost and most sloping roots of the plexus. Sometimes the fifth cervical nerve is literally torn across at the intervertebral foramen, but more often its fibres are ruptured within the nerve sheath at the point where the fifth and sixth nerves join together, and frequently scar tissue can be seen surrounding the two nerves at this point. The paralysis resulting from this lesion is immediate, and involves the deltoid, supra- and infraspinatus, the biceps and brachialis anticus, and supinator longus muscles, with resultant inability to abduct the arm from the side or to flex the forearm at the elbow. The arm therefore appears extended, adducted to the side, and at the same time rotated inwards, subsequent wasting of the deltoid causing flattening of the usual rounded appearance of the shoulder, with prominence of the acromion process. Other muscles that also may be paralysed in some of these cases are the subscapularis, the radial extensors of the wrist, and pronator radii teres, though the paralysis of these latter muscles causes no obvious great disability, as their action is partly compensated for by others. Usually there is no loss of sensation whatever on the arm or hand, which is due to the fact that the fifth cervical nerve contains fewer sensory fibres in proportion than the other nerves entering the plexus. When the sixth cervical nerve is also badly damaged, slight anæsthesia of the thumb and outer side of the hand may be detected. This form of injury is also found in new-born infants in whom traction by the hand or by a hook has been exerted ; it is then known as Erb-Duchenne paralysis. The slighter cases of this type of injury may recover, particularly if aided by adequate massage and electrical treatment, but many of the severer forms give rise to permanent paralysis, which shows no improvement whatever under massage or electricity, the affected muscles wasting completely. In these cases the rupture of the nerve-fibres in the sheath is complete, and the formation of scar tissue prevents the re-establishment of the continuity of the nerve by the down-growing nerve-fibrils from the central end above the injury. When this is the case the question of treatment by nerve suture will arise, and the records show many cases in which improvement or even cure has resulted from the cutting away of the scar tissue found at the junction of the fifth and sixth cervical nerves, and the suture of the central and peripheral ends of the nerves again together.

Injuries of the first dorsal and eighth cervical nerves by forcible traction of the arm have been known as Klumpke's paralysis, the muscular atrophy and paralysis in these cases affecting the intrinsic musculature of the hand, and to a less extent the flexors of the fingers, but in this case anæsthesia will be a prominent feature, practically corresponding to the ulnar and internal cutaneous nerve distribution on the inside of the hand and forearm. In some of these cases, moreover, when the first dorsal nerve has been injured close to the intervertebral foramen, there will be signs of cervical sympathetic paralysis upon the same side, the pupils being small and not dilating properly when shaded, and the palpebral fissure slightly narrowed by drooping of the upper lid. In cases of extreme violence the whole attachments of the brachial plexus and its spinal nerves may be torn out of their intervertebral foramina, or even from the spinal cord itself. In the latter case hæmorrhage may take place into the spinal cord, and varying degrees of paralysis of the lower extremities of a spastic type may result, in addition to the total flaccid paralysis of the upper limb.

Cervical Rib

Owing to the upward curve of the first dorsal nerve over the first rib on its way to join the eighth cervical nerve, this lower nerve is particularly liable to injury from violent traction downwards of the limb, and in those cases of abnormality in which a cervical rib attached to the seventh cervical vertebra is present, the first dorsal nerve will have a much more acute bend in its course, having to pass over this rib, or the ligament connecting its tip to the first rib, on its way to join the plexus. In early adult life, therefore, various symptoms such as tinglings and pain down the inside of the forearm and hand, with anæsthesia of the inside of the forearm down to the wrist, and wasting of the intrinsic muscles of the hand, may gradually develop, their origin sometimes dating from, or perhaps being accentuated by, an injury.

During the passage of the nerves through the plexus they may be injured above the clavicle by heavy blows from falling weights, stab or bullet wounds, or by prolonged pressure as from the carrying of a heavy sharp-cornered box. The two nerves which are particularly subject to pressure, or from blows by heavy weights, are the long thoracic or nerve to the serratus magnus, and the posterior scapular nerve supplying the rhomboids and the levator anguli scapulæ. These two nerves, moreover, may be damaged through violent contraction of the scalenus medius muscle, and the spinal accessory or nerve to the trapezius may easily be severed by a punctured wound outside the middle of the sternomastoid muscle. Indeed, isolated paralysis of one trapezius muscle, partial or complete, is perhaps the commonest local muscular paralysis met with about the shoulder-girdle. In addition to its liability to suffer from injury to the spinal accessory nerve by stab or bullet wounds, neuritis from pressure or exposure to chill or infection, the nerve is often involved by tubercular or other forms of adenitis in the posterior triangle, and is especially liable to be cut in operations upon these glands.

Paralysis of the trapezius will result in obvious deformity of the shoulder-girdle, the wasting of its upper thick portion rendering the clavicle obviously visible over the top of the scapula when seen from behind. The vertebral border of the scapula falls away from the middle line, especially at its upper angle, and the whole scapula will be tilted upwards by the pull of the levator anguli scapulæ. The arm is not very much weakened as a result of this paralysis, except in the work of carrying weights upon the shoulder. Paralysis of the rhomboids and levator anguli scapulæ from a traumatic neuritis of the **posterior scapular nerve** may result from a sudden excessive effort, as in heaving earth upwards with a spade out of a grave. Sudden sharp pain will be felt in the region of the top of the scapula and down its vertebral border, where some tenderness will be developed. Slight winging of the scapula and lowering of its inferior angle results from this paralysis, and pressure downwards with the elbow against resistance will cause the inferior angle of the scapula to slip outwards, owing to the loss of the resultant pull of the rhomboids.

Paralysis of the **serratus magnus** is one of the commonest of these isolated nerve paralyses, the long thoracic nerve being damaged by pressure in the posterior triangle, as in carrying a box upon the shoulder, or it may be damaged by violent contraction of the scalenus medius through which it passes.

Serratus magnus palsy gives rise to a very marked winging of the scapula when the arm is held out in front, and there is also inability to raise the arm above the horizontal, owing to the loss of the normal action of the serratus muscle in rotating the scapula.

Wounds of the nerve trunks in the neck above the clavicle, such as by stab or bullet wound, may involve either the posterior or the outer cords of the plexus, the inner cord usually escaping, and numerous instances have occurred in the present war from bullet wounds traversing the top of the chest either just below or just above the clavicle. The posterior cord appears to be by far the trunk most frequently damaged. In some cases not the whole cord may be cut, but only the branch going to it from one or more of the main spinal nerves. Thus I have seen complete wrist-drop and other symptoms of **posterior cord paralysis** result from a bullet wound which grooved the upper edge of the clavicle about its middle, and cut across the branch to the posterior cord given off from the junction of the fifth and sixth cervical nerves. Although the branches to the posterior cord from the seventh and eighth cervical nerves were not injured in this case, yet all the musculature supplied by the posterior cord was paralysed, with full reaction by degeneration, the only difference detectable from a complete paralysis of the cord being that the anæsthesia of the outer side of the back of the hand and forearm was incomplete. Further, recovery of voluntary power of the paralysed muscles in this case, after suture of the divided branch, was more rapid than would be expected if the whole posterior cord had been divided, the improvement in power being well advanced three and a half months after the operation. The paralysed muscles in such a case comprise the latissimus dorsi, the deltoid, the teres major, subscapularis, the triceps and all the extensors of the wrist and digits, and the supinators.

Sensation is diminished to touch and to pin-prick over the outer side of the back of the hand and forearm. Injury to the **outer cord** will produce paralysis of the flexors of the forearm and some weakness of the pectorals, and in addition certain muscles of the flexor mass in the forearm deriving their nerve supply through the outer head of the median nerve, the flexor carpi radialis, and the pronator radii teres. There will be also anæsthesia along the outer side of the front of the forearm supplied by the musculo-cutaneous nerve, and most of the median nerve distribution on the palm of the hand, the thumb, and the two outer fingers. The **inner cord** lies more deeply protected by the clavicle, and is in close approximation to the subclavian artery and vein, and punctured wounds involving the inner cord will be likely to produce fatal hæmorrhage. If the patient escapes this latter event, injury to the inner cord will cause wasting of all the muscles in the hand with paralysis of flexion of the two inner fingers and of the flexor carpi ulnaris, together with anæsthesia of the inside of the arm and forearm and hand corresponding to the cutaneous supply of the intercosto-humeral, the two internal cutaneous nerves, and the ulnar nerve. The motor paralysis of the inner cord lesion differs from that due to ulnar nerve lesion, in that there is additional paralysis of the muscles supplied through the inner head of the median nerve.

Dislocation of the Shoulder is a frequent cause of severe injury of the upper extremity. The patient falling with the arm outwards commonly produces a downward dislocation of the head of the humerus, which frequently causes immediate paralysis of the deltoid and the infraspinatus, through the sudden stretching of the deep motor branch of the circumflex nerve which winds round the head of the humerus to enter the under-surface of the deltoid, while the infraspinatus is paralysed owing to the sudden stretching of the suprascapular nerve due to the sudden pulling downwards of the infraspinatus attachment to the greater tuberosity of the humerus. Although the dislocation of the shoulder may be immediately reduced, yet the paralysis and subsequent wasting of the deltoid and of the infraspinatus muscles continues, the lesion thus resembling a partial Erb's paralysis. In a few of these cases there is slight anæsthesia of the skin over the deltoid, owing to the involvement of the cutaneous branch of the circumflex nerve.

The inward pressure of the dislocated head of the humerus may produce severe traumatic neuritis of the main nerve trunks issuing from the brachial plexus, namely, the musculo-spiral, musculo-cutaneous, median, and ulnar nerves. Of these the musculo-spiral is the one most usually affected, though all combinations and degrees of paralysis are seen in different cases.

Musculo-spiral paralysis is one of the commonest of the nerve injuries of the upper extremity, and this nerve is frequently divided by bullet and stab wounds, or it may be torn by the fragments of a fractured humerus, or become involved later in the callus, or in the scar of a septic wound. Pressure upon the musculo-spiral nerve as it winds round the humerus is one of the commonest causes of its injury, as in crutch palsy or in sleep palsy, when the nerve may be injured by resting the arm over the sharp edge of a wooden chair, or by the pressure of the head upon the arm. Musculo-spiral paralysis will resemble posterior cord paralysis in the production of dropped wrist, and paralysis of all the extensors supplied by the posterior interosseous nerve, and anæsthesia of the back of the outer side of the hand supplied by the radial nerve. The triceps muscle will be paralysed if the lesion is high up, above the exit of the nerves to the triceps, as in crutch paralysis, though in sleep paralysis and in fractures of the humerus when the nerve is involved this muscle usually escapes. There is also paralysis of the supinator longus in all cases of musculo-spiral paralysis, a fact which is of considerable value in the differential diagnosis from an hysterical paralysis, or from a dropped wrist due to lead poisoning.

In hysterical paralysis of extension of the hand, or in malingering of this condition, flexion of the forearm may be perfectly well carried out, and in those cases the supinator longus will be seen to act strongly with the biceps in flexing the forearm. It should be remembered, however, that paralysis of the posterior interosseous nerve by injuries at or below the elbow will not affect the supinator longus muscle or the long radial extensor of the wrist, the paralysis in these cases affecting the extensors of the thumb and fingers and the ulnar extensor of the wrist. This is not a very uncommon injury from stab wounds or shell and bullet wounds in the forearm. In these cases of injury to the posterior interosseous nerve there will be no cutaneous anæsthesia, owing to the radial nerve having been given off from the musculo-spiral nerve just above the elbow.

A fairly common cause of musculo-spiral neuritis is from compression of the nerve in the upper arm by violent contraction of the triceps muscle. This may occur during the delivering of a violent blow with the arm as in boxing, or in the throwing of a stone or cricket-ball, or it may be compressed and damaged during the lifting of a heavy object with the arms extended, as in the carrying of heavy flower-pots, or in lifting a heavy patient off an operating-table. As a result, a perineuritis involving the sheath of the musculo-spiral nerve is produced, which gives rise to severe aching pain down the arm to the back of the wrist. This pain will closely resemble that of sciatica, and there is a close analogy between musculo-spiral neuritis produced in this way and sciatic neuritis, which may be started by a blow or by a violent muscular effort (see page 88). The damage of the nerve is not severe enough to produce paralysis and dropped wrist; indeed motor weakness, as in sciatica, is usually absent. Movements of the limb, however, produce pain, especially movements involving leaning down with the arms stretched out, as in lacing up the boots. There will be some tenderness of the nerve trunk on pressure where it winds round the humerus a few inches above the elbow, and tingling sensation will be produced in the back of the forearm and wrist.

In the majority of cases no anæsthesia is to be detected.

The treatment, as in the case of the more extensive inflammation of brachial neuritis, will necessitate rest of the limb, and moist heat in the form of poultices. Anodyne liniments may give a certain amount of relief, and analgesic drugs, such as aceto-salicylic acid, phenazone, &c., are usually of service in allaying the pain, though sometimes morphia may also be necessary to produce sleep.

In the treatment of dropped wrist from musculo-spiral paralysis it is an important point not to permit the wrist to hang down continually in the dropped position, inasmuch as paralysed muscles will remain paralysed almost indefinitely so long as they are hyperextended. Some apparatus therefore must be used to keep the wrist extended. Many practitioners adopt some form of splint for this purpose, though I dislike this method, as I have seen it often produce considerable injury and fixation of the wrist-joint. A fairly efficient and light apparatus consists of an ordinary leather wrist-strap, and a broad strong tape fastened around the bases of the three inner fingers, the tape being attached to the back of the wrist-strap by means of a piece of broad strong elastic. The tension of the elastic can be graduated and will be usually sufficient to keep the wrist from being overflexed.

Musculo-cutaneous injury produces paralysis of the flexors of the forearm, with the exception of the supinator longus and a small portion of the brachialis anticus, which are supplied by the musculo-spiral nerve; anæsthesia of the outer side of the flexor aspect of the forearm, supplied by the cutaneous branch, will also be present.

This nerve is only occasionally damaged by the pressure of the head of the humerus in dislocations of the shoulder, though it is liable to suffer from bullet and stab wounds of the front of the upper arm.

Median Nerve Paralysis

This nerve occasionally suffers severely from compression by the head of the humerus after dislocation of the shoulder-joint. The nerve gives off no branches until it reaches the elbow, and lying in close proximity to the brachial artery and its attendant veins on the inner side of the arm, it is not exposed to injury from fracture of the humerus as is the musculo-spiral nerve. Stab or bullet wounds may, however, damage the nerve with or without damage to the main blood-vessels. The result of median nerve paralysis is loss of power of flexion of the thumb and of the index-finger completely, though the flexion of the two inner fingers will still be fairly powerful through the ulnar supply to the flexor profundus digitorum. The thenar eminence and the finger pads of the thumb and two outer fingers will atrophy, and cutaneous anæsthesia will be found over the palmar surface of the hand, with the exception of the little finger and inner half of the ring-finger, and on the dorsum of the index, middle, and outer half of the ring-finger as high as the first interphalangeal joints. After several weeks' paralysis the skin becomes thin and shiny, what is known as 'glossy skin', and peri-articular adhesions are liable to form around all the interpharyngeal joints. This latter condition is insidious in its onset, and may be recognized by the pain produced on bending the fingers. Treatment of the condition must be directed towards preserving the mobility of the joints by massage and moist heat. The median nerve sometimes suffers in the forearm to a less extent by prolonged pressure, as in leaning the flexor surface of the forearm upon the sharp edge of a desk. In this case, as a rule, the only muscles paralysed are the thenar eminence and the two outer lumbricals, and light anæsthesia and paræsthesiæ of the median nerve distribution will accompany the motor paralysis. The median nerve is very liable to injuries at the wrist from cuts by knives or by broken glass, a particularly common accident being the severing of tendons and of the median or ulnar nerve at the wrist, by pushing the hand through a glass window. In all such injuries of cut tendons it is therefore highly important, before suturing the tendons, carefully to examine the fingers for anæsthesia of the median or ulnar distribution, as primary suture of the cut nerve is far easier to do at the time than afterwards, and the chances of recovery of the damaged nerve are also much enhanced. A mistake which I have known made is to suture the cut nerve to a tendon, but such a mistake need only be remembered to be avoided.

Occupation neuritis not infrequently affects the muscles of the thenar eminence supplied by the median nerve, and atrophy of the opponens and abductor pollicis, usually without any anæsthesia, is a chronic condition sometimes met with in scrubbers and gardeners, &c. This is a particularly obstinate form of chronic atrophy, and rarely improves with treatment. Pressure in the palm of the hand through leaning heavily on the knob of a walking-stick is also liable to cause persistent tingling and light anæsthesia, due to chronic neuritis of the median nerve branches in the palm being set up. Any of these branches may be damaged, moreover, by penetrating wounds of the palm of the hand.

Ulnar Nerve Paralysis

This nerve also may suffer from pressure at the elbow, where the nerve runs in a groove on the back of the inner condyle of the humerus. When the elbow is acutely flexed the nerve is extended in its groove, and this extension, combined with pressure through prolonged leaning upon the elbow, may cause pressure paralysis and a degenerative neuritis. Blows upon the elbow, fractures of the lower end of the humerus, and bullet or stab wounds are frequent causes of injury of the ulnar nerve near the elbow-joint. The nerve is comparatively rarely injured in the middle and upper portions of the arm, owing to its somewhat sheltered position below and behind the median nerve and the brachial artery. Like the median nerve, it is occasionally damaged by pressure of the head of the humerus after dislocation of the shoulder-joint. As the ulnar nerve gives off no branches above the elbow-joint, symptoms produced by its paralysis will not vary with the position of its injury in the upper arm. There will be paralysis of the ulnar flexor of the wrist and of the inner half of the flexor profundus digitorum, though owing to the median nerve supply of the flexor sublimis digitorum there will be very little noticeable weakness of flexion of the fingers, and it will be found on testing that the flexion of the little finger is weak and easily overcome. In addition there will be paralysis and subsequent wasting of the hypothenar muscles and interossei and of the adductor of the thumb, with resulting claw hand, the hyperextension of the first phalanges and flexion of the second and third being more marked with the ring and little fingers, owing to the escape of the two outer lumbricals which are supplied by the median nerve. Anæsthesia in cases of division or complete paralysis of the ulnar nerve will be total to all forms of sensation on the little finger, and cutaneous anæsthesia to touch and pin-prick will be practically complete over the inner side of the hand up as high as the wrist, and the ulnar side of the ring-finger, though deep pressure may still be perceived. Owing to the subsequent wasting of the interossei there will be marked hollows between the metacarpal bones, and this is particularly evident between the metacarpal bones of the thumb and index-finger, due to wasting of the first dorsal interosseous muscle. Owing to the paralysis of this muscle the index-finger will be turned over towards the ulnar side of the hand, and it cannot be abducted towards the thumb. As a rule there is little or no glossy skin noticeable in ulnar nerve lesions, nor will there be any periarticular adhesions of the finger-joints. With lesions about the level of the wrist-joint, as from slashes across the wrist with a knife, or from broken glass, the paralysis of the hypothenar and interossei muscles will be exactly the same as when the nerve is injured higher up, though there will be no weakness now of the flexors.

When the ulnar nerve is injured close to the wrist-joint, the inner border of the back of the hand will escape anæsthesia, owing to the dorsal cutaneous branch being given off from the ulnar nerve above this point, though the little finger will be anæsthetic as in the former case. The deep motor branch of the ulnar nerve in the hand may be injured alone after it has separated from the cutaneous portion, by a punctured wound of the hypothenar eminence, as by a fall upon the hand on a piece of broken glass or the point of a knife. In such a case the interossei and the two inner lumbricals will be paralysed, and will waste without any loss of sensation on the little finger. It is very important when suturing wounds of the flexor aspect of the wrist to make sure of the condition of the ulnar nerve, if the wound is in its neighbourhood, as in many cases cut tendons have been picked up and sutured, though the divided ulnar nerve has been overlooked. If the wound suppurates, the damage to the nerve will be much greater, as it is liable to become buried in dense scar tissue.

The difficulties of treating this condition are great, as in operations undertaken to free the nerve from the scar tissue it may be necessary to cut away 1 to 1½ inches of the nerve. It will then be almost impossible to bring the two ends of the nerve together, though this may be sometimes done by strong flexion of the wrist, and fixing the wrist in this position by a splint for a few days. The danger of the reinclusion of the nerve in scar-tissue is great, though this may be avoided by wrapping the nerve in a piece of fascia freshly dissected from the thigh, or in a piece of cargile membrane. It is very important that the patient should regain the sensation supplied by the ulnar nerve, as a hand with ulnar nerve anæsthesia will be quite useless for any delicate work. In a not inconsiderable number of cases it will be found that after primary or secondary suture of a divided ulnar nerve, sensation reappears gradually after some months, and may become practically completely re-established, though the interosseal wasting and clawed hand do not recover. Though this is somewhat unsightly, yet the hand will be a very useful member, far more so than if the anæsthesia had remained permanent and the motor paralysis had been recovered from.

Ascending Neuritis

Septic injuries of a nerve trunk or branch occasionally give rise to a spreading neuritis which ultimately affects other branches arising from the same nerve trunk higher up. Thus I have seen a septic injury of the thenar eminence followed later by wasting of the flexor mass in the forearm supplied by the median nerve, with anæsthesia and glossy skin of the thumb and index-finger, accompanied by persistent pain. Similarly, I have seen slight septic infection of an operation wound in the neck after nerve suture had been performed, followed by gradual spreading of septic infection throughout the whole brachial plexus, constant pain and tenderness of the whole upper extremity being produced, with glossy skin and peri-articular adhesions of the finger-joints. In certain rare cases ascending neuritis may spread upwards along the spinal roots to reach the spinal cord, thus setting up myelitis.

This is apparently rare with the nerves of the upper extremity, but I have known three or four instances of this condition affecting the lumbo-sacral plexus. Thus, prolonged perineal pressure from a bicycle saddle has not infrequently caused anæsthesia of the urethra, and even of the bowel, with temporary anæsthesia and incontinence of the sphincters, and this condition has been known to be followed by a sacral myelitis and death. Ischio-rectal abscess has also been known to give rise to an ascending neuritis followed by sacral myelitis. Chronic sciatica, too, I have known to be followed by an acute sacral myelitis after too energetic and prolonged vibratory massage, which caused severe pain after each application.

Brachial neuritis as the result of injury has already been partially dealt with when describing the results of nerve injuries after dislocation of the shoulder. An acute inflammation of the main trunks of the brachial plexus may, however, be produced as a result of a fall upon the shoulder, or severe wrenching of the arm without any actual dislocation of a joint or fracture being produced. There will be severe and constant pain usually radiating from the point of the shoulder along the outer side of the arm, down the back of the forearm into the wrist and fingers. Though the pain is more or less constant and associated with considerable tenderness of the whole limb, the pain will vary in its intensity at different times, being often particularly severe at night, and any movement of the limb, even slight joltings or vibrations of its supports, will intensify the suffering. Considerable swelling of the whole limb and œdema of the back of the hand are sometimes, but by no means always, seen. Such œdema is neurotrophic and vasomotor in its origin, and is not necessarily associated with any obstruction of the venous return in the arm. Usually there is no anæsthesia of the skin, but on the contrary, some hyperæsthesia.

The treatment of this condition necessitates complete rest to the upper extremity, which should be surrounded with cotton-wool, very lightly bandaged on, and the limb supported in a sling against the side. The patient is best kept in bed or on a couch, when the limb may be supported by pillows. Various local treatments are sometimes of considerable value in allaying the pain, which is often very intense. A liniment made of equal parts of the aconite, belladonna, and chloroform liniments (A.B.C.) should be made hot and sprinkled over warmed cotton-wool applied around the arm and neck, and covered with a thick layer of dry cotton-wool and a light domett bandage. This may be changed every four hours, though the arm should be moved as little as possible in doing so. Methyl salicylate and menthol liniments give more relief in some cases, though they must be applied with the utmost gentleness, and nothing in the nature of massage must be used.

Antiphlogistin poultices applied hot and kept on for twenty-four hours are sometimes very soothing, and these may be imitated by using a mixture of china clay and glycerine with menthol. Radiant heat applied locally by incandescent lamps with reflectors twice a day is a useful help before changing the anodyne liniment. In spite of these remedies the pain is sometimes so severe that injections of morphia will be necessary, together with a nightly draught of bromide, of veronal, or some similar hypnotic. Without the morphia the hypnotic is useless in cases of severe pain. In the less severe cases, or when improvement is commencing and the tenderness is less intense, treatment by salicyl ionization may aid recovery, a large thick pad of Gamgee tissue soaked with warm 2 per cent salicylate of soda solution being applied over the shoulder and the side of the neck, in connexion with the negative electrode of a galvanic battery. The positive electrode should be in similar connexion with a thick pad soaked with lithium carbonate solution, wrapped around the forearm. Twenty minutes' treatment twice a day with a moderately strong current should be employed, and its effect noted. If the pain is aggravated it should at once be given up.

When hyperæsthesia of the upper extremity is very intense, a point that should never be lost sight of is the possibility of the whole condition being hysterical and not due to an actual brachial neuritis. A somewhat similar condition is seen in the **painful stump** of hysterical origin which may follow amputation. The hysterical cases are usually to be distinguished from true painful neuroma by the exaggerated condition of hyperæsthesia present, affecting large areas of skin, which should not be tender in neuroma. Any movement, moreover, is likely to cause apparent pain in the hysterical painful stump. Inconsistence of the patient with regard to his pain will usually give away his case. Thus I have seen a man, who had amputation performed through the middle of his forearm on account of an injury, develop intense pain in the stump a week afterwards, the flap being opened and explored by the surgeon after a fortnight. Nothing was found to account for this pain, and he was sent out of hospital, being told to wear his arm in a sling and protect it carefully. A fortnight later this man walked up from Maidenhead twenty-six miles to St. Mary's Hospital, with his arm swathed in cotton-wool at least two inches thick, and carrying his arm in a sling on account of the intense tenderness any touch or movement provoked, as he said. Yet the very fact of his being able to walk with his arm in that condition proved beyond a possibility of doubt that the tenderness was psychical and not physical. After unwrapping his limb nothing abnormal was found, though the man complained of intense hyperæsthesia for any touch or movement, However, firm treatment with faradism and the wire brush, combined with suggestive encouragement, dissipated the hyperæsthesia entirely and cured him of his trouble in less than five minutes.

Ischæmic Myositis and Neuritis

Ischæmic myositis, or Volkmann's contracture, was described many years ago as the result of prolonged tight bandaging of a limb. This condition is almost limited to the muscles of the forearm, and is generally produced as the result of the limb being bandaged too tightly upon a splint, after fracture of one or both bones of the forearm. A splint, however, is not a necessary factor in its production, and I have seen ischæmic myositis produced by bandaging the forearm tightly for a sprained wrist. If the bandage is very tight, and severely obstructs the circulation of the blood and lymph for twelve hours and more, there is likely to be set up a form of primary contracture in the muscles resembling rigor mortis, with the exception that the contracture is permanent. The flexor muscles of the fingers are those usually involved most, and it will be found on taking off the splint and bandage that the fingers cannot be straightened.

Previous to this stage the patient will have complained of considerable pain in the hand and fingers, which become bluish and discoloured. It has been usually stated that ischæmic myositis is purely a muscular condition, and that there is no reaction of degeneration, the muscles still reacting normally to faradism. My own experience, however, of a number of these cases has shown in the majority undoubted evidence of accompanying neuritis, of the ulnar or median nerves or both, with cutaneous anæsthesia, wasting of the finger-pads, periarticular adhesions, and well-marked reaction of degeneration in the thenar muscles, and sometimes in the flexors of the fingers. Occasionally, ischæmic myositis is present alone without neuritis, in which case the faradic reactions of the affected muscles are normal, though in most cases I have found neuritis also present, which is not to be wondered at, seeing that the nerves are exposed equally with the muscles to damage from the prolonged tight bandaging.

The treatment of this condition is very difficult. Though the neuritis will recover in time, the primary muscular contracture is permanent, and can only be remedied by some surgical procedure for lengthening of the tendons, as massage, even if long continued, seems to make little or no impression on the damaged tissue.

Traumatic Neuro-Fibrositis

Muscular strains of the upper extremity, particularly in the neighbourhood of the scapula, are liable in certain persons, more especially if there is a gouty or rheumatic family tendency, to be followed by chronic tenderness and pain on movement, which may persist for months, or even years. The site of the tenderness and pain is usually between the vertical border of the scapula and spine. The pain is, however, not local only, but is usually referred up the back of the neck and over the shoulder-joint, running down the arm even into the fingers. Pressure internal to the vertebral border of the scapula will elicit areas of intense tenderness, causing radiating neuralgia similar in character and extent to the spontaneous pains, even though the tender point, pressure on which elicits the pain down the arm, may be as low in the back as the inferior angle of the scapula, far below the distribution of the brachial plexus. Sometimes these tender points, and the pain associated with them, are too sensitive for even light massage to be borne, much less the deeper searching of Swedish manipulators, and all forms of local treatment, such as radiant heat, ionization, anodyne liniments, and poulticing, may fail to give relief.

In many cases where the tender points are very localized and definite, I have seen rapid cure produced by local injection of the tender spots with strong alcohol. This should be done with a fine needle plunged into the centre of the spot down to the bone, rib or scapula as the case may be. A few drops of 2 per cent novocain solution are injected, and after allowing a minute for the anæsthetic effect of the drug to take place, a fresh syringe containing

90 per cent alcohol is fitted on to the needle, which has been kept in position all the time, and ten minims of the spirit are injected. This will give rise to some burning pain, which may be fairly sharp for ten or fifteen seconds, and then rapidly dies away, though again during the night there may be considerable aching pain requiring aspirin and phenacetin, or an injection of morphia. Sometimes the relief from pain is immediate and permanent, though in others several spots may require treatment before a cure is effected.

Traumatic neuro-fibrositis is perhaps even commoner in the lumbo-sacral regions, and may complicate a sciatica, or may be mistaken for this complaint (see p. 91). If the pain is severe on movement, complete rest in bed must be tried at first, with local treatment by fomentations and glycerine of belladonna and laudanum, alternated with local applications of radiant heat. Sometimes massage is very useful, but it must be used tentatively at first, as if the pain is increased after the first two treatments, the massage must not be persisted in. Hot brine packs, or better daily soaking in strong brine baths at Droitwich, sometimes bring about complete relief. In other cases, ionization with salicylate of soda daily for twenty minutes may give good results. Some practitioners have found specially good results by using the arc lamp as the source of the radiant heat, and electrical treatment by the static induced current or by X-rays has also had good results.

CHAPTER IV

INJURIES OF THE LOWER EXTREMITY

THE nerves in the lower extremity which are particularly liable to injury are the anterior crural nerve and the sciatic nerve and its branches. The **anterior crural nerve** derives its nerve supply from the second, third, and fourth lumbar nerves, and passing over the surface of the ilio-psoas muscle, enters the front of the thigh underneath Poupart's ligament on the outer side of the femoral vessels. It will, therefore, be liable to injury from penetrating wounds by knife or bayonet thrust, shell and bullet wounds, &c., in this region, and either total or partial paralysis of this nerve may be produced by such injuries, or a traumatic neuritis of the anterior crural nerve may be set up by injuries in its neighbourhood, particularly if there is suppuration of an open wound. Scar tissue will then be formed in considerable quantity, and irritation of the nerve may be kept up indefinitely. Anterior crural nerve injury produces paralysis and wasting of the quadriceps extensor muscle, so that there will be inability to extend the leg at the knee-joint, and there will be marked atrophy of the muscle mass on the extensor surface of the femur. Reaction of degeneration will be found in this muscle, and the knee-jerk will be entirely lost. Cutaneous anæsthesia to touch and pin-prick will be present over the dorsum and inner side of the thigh down to the level of the patella, and of the distribution of the long saphenous nerve down to the inner side of the foot. Portions of the anterior crural nerve may be damaged by local injuries to the front of the thigh, so that cutaneous anæsthesia may result without any muscular paralysis. The treatment of anterior crural paralysis will depend upon the form of injury, and if total extensor paralysis is present with anæsthesia, followed by muscular atrophy, the indication is that the nerve has been severely damaged, perhaps cut across, and exploration by operation will be advisable with the idea of resuturing the cut ends of the nerve. Massage and galvanism should also be employed to limit the muscular atrophy as far as possible.

Owing to the paralysis of the quadriceps extensor cruris, the gait is somewhat weakened and the patient will be unable to run. In walking, the body will be rolled over to the affected side when coming forwards on to that foot, in order to get the centre of gravity directly over the leg.

The Obturator Nerve, owing to the depth of its exit from the pelvis into the thigh through the obturator foramen, is rarely injured, though it may be severed by a bullet wound, and crushing of the nerve has been known to occur in cases of difficult labour. Paralysis and wasting of the adductor muscles of the thigh will ensue in such a case. The operation for nerve suture, though just possible in the case of the anterior crural nerve, is practically impossible of accomplishment in cases of division of the obturator nerve.

SCIATIC PARALYSIS

The **Sciatic Nerve** is frequently injured after its exit from the pelvis at the great sciatic notch. This nerve, the largest in the body, is about three-quarters of an inch in width, and is somewhat loosely built in structure, and is not a firm cylindrical cord like the main nerve trunks of the upper extremity. It is composed of the fifth lumbar and first and second sacral ventral branches, which after passing through the psoas muscle unite to form a thick cord, which passes posteriorly into the buttock through the great sciatic notch. The small sciatic nerve, which also issues from the sacral plexus by the great sciatic notch, and which supplies the gluteus maximus, and also the skin of the back of the thigh and calf with sensation, may be considered as one nerve with the great sciatic clinically. Though protected in this region by the thick mass of the glutei, the sciatic nerve is here not infrequently injured through muscle strains and subsequent fibrositis, causing perineuritis of the sciatic sheath. Injuries from penetrating wounds in the region of the buttock are not very rare, and the nerve may be totally divided close to the notch by a bullet wound or thrust from a bayonet. As a result there will be paralysis and wasting of the gluteus maximus if the small sciatic, with its inferior gluteal branch, be also damaged, and of the hamstrings, and particularly of all the muscles in the leg and foot below the knee, with reaction of degeneration of all these muscles to electrical testing. Anæsthesia of the foot and leg up to the knee will be present, with the exception of the area supplied by the internal saphenous nerve along the side of the foot and toes. Loss of sensation will also be found in the distribution of the small sciatic nerve on the back of the calf and thigh up to the level of the inferior gluteal fold. Owing to the paralysis of the muscles of the leg below the knee, there will be dropping of the foot, which will be dragged in walking. Trophic sores occasionally develop as the results of slight damage, or from pressure on the outer surface of the foot or leg, but these are by no means constant, and usually yield to ordinary antiseptic treatment. In cases where the sciatic paralysis has resulted from a penetrating wound, as in cases of gunshot injury, operation will be necessary to explore the site of the injury in order to suture the cut ends of the nerve. These are likely to be found buried in firm scar-tissue, if the operation is delayed, as it should be, until the wound of injury is healed and all suppuration has disappeared.

Bullet wounds through the lower part of the thigh may chance to damage either the external or the internal halves of the sciatic separately. I have seen a case where division of the two nerves took place higher up in the thigh than usual, in which a bullet wound just above the back of the knee cut through both nerves, though separated from one another. In this case some of the extensors of the foot escaped paralysis owing to the injury to the external popliteal nerve being below the point of the exit of their branches.

It is dangerous to expose a nerve to operation while there is still danger of local infection of the nerve from a septic wound. Scar tissue will appear to unite the cut ends of the nerve, so that the nerve may almost appear continuous, but close examination and faradic stimulation with a sterilized electrode will demonstrate the absence of true nervous continuity through the scar. It will be necessary boldly to cut out the scar tissue and to resuture the cut ends of the nerve. It should be remembered, and the patient also should be warned, that no signs of recovery are to be expected until at least three months have elapsed, and that it will very likely be six months before definite signs of improvement in sensation have been demonstrated over the previously anæsthetic area. Somewhat earlier than this, however—perhaps after three months—the patient may be conscious of some improvement, and though there be no apparent return of voluntary power in the foot, and the anæsthesia may appear the same as before, yet the patient will feel that the leg seems to be more alive and to be less helpless than formerly. As soon as the wound of operation for the suture of the damaged nerve has healed, treatment of the paralysed limb by massage and by interrupted galvanism must be commenced. One electrode should be wrapped round the ankle, and the other should be applied underneath the back, the patient lying upon it, and should not be applied over the buttock for some weeks after the operation, owing to tenderness of the scar. A galvanic battery should be used, and turning on enough cells to show a current of 8 to 10 milliampères on the galvanometer, the current should then be reversed rhythmically with the current reverser about once per second. This may be either worked by hand, or an automatic clockwork current reverser of the metronome type, fitted with a small Pohl's commutator, may be employed.

The treatment with the galvanic current should thus be employed twice a day for about twenty minutes for months, until either the voluntary power of the muscles of the foot or the faradic excitability is beginning to return. When this is the case, the faradic current may be substituted for the galvanic, and should be applied either by a roller electrode, or the method of wave faradism should be used, the electrodes being applied as described above, and the sledge coil moved backwards and forwards in order to strengthen and diminish the intensity of the current rhythmically, the buzzer being kept vibrating continuously. This method is rather wasteful of the battery current of the cells supplying the coil, and they will not last nearly so long by this method as if the interruptor were made to vibrate slowly at the rate of about twice per second. This can either be done by working the interruptor spring by hand, or by fitting on to the interruptor hammer a weight arranged to make it vibrate more slowly. This is only possible with certain types of battery. The advantage of the slow faradic shocks by this method is to allow the muscles to relax after the momentary contraction produced by the single faradic shock, and the treatment by wave faradism has the same general effect of allowing relaxation of the muscles after a series of rapid tetanic contractions. Relaxation of a muscle is physiologically as important as its contraction, and if the muscle be kept strongly tetanized for twenty minutes continuously by the faradic current, rapidly interrupted, the effect is likely to be harmful and not beneficial.

Pressure paralysis of the sciatic nerve high up in the buttock is not commonly met with, though it is sometimes seen as a result of lying upon a hard surface for many hours during coma or other conditions producing total inability to move. A number of cases of sciatic paralysis have occurred in this way in coal-miners after a gas explosion, in whom prolonged coma for twenty-four hours or longer has been produced by inhalation of the carbon monoxide and dioxide gases. Lying in one position upon the hard and rough floor for many hours without moving may then produce a pressure paralysis of the sciatic nerve near its exit from the sciatic notch, of varying degrees of severity. The same result may happen on the battlefield from prolonged pressure upon the nerve in the case of a wounded soldier left out in the open for one or more days.

Puerperal paralysis of the sciatic nerve is occasionally the result of a difficult labour, in which forceps have been used, the nerve being probably pressed on by the forceps during traction. As a result, there will be severe foot-drop and muscular wasting of the leg, with reaction of degeneration. In some cases the paralysis is limited to the peroneal distribution, while in others the internal popliteal suffers also.

Pressure paralysis is a not uncommon effect upon the external popliteal nerve from pressure produced in various ways. For instance, operations in the Trendelenburg position, or other positions necessitating the fixation of the legs by clamps below the knee, may produce peroneal paralysis on one or both sides by the prolonged pressure of the clamps upon the peroneal or external popliteal nerve. The resultant paralysis will be dropped foot and inability to dorsiflex the ankle or to extend the toes, or to evert the foot by the peronei muscles. Varying degrees of numbness and anæsthesia will be produced, affecting the dorsum of the foot and the outside of the lower three-quarters of the leg. After the lapse of a week, reaction of degeneration will appear in the tibialis anticus and all the extensors of the toes and the peronei, the limb will be bluish owing to vasomotor paralysis, and its superficial temperature will be diminished and the patient will complain of the leg and foot feeling cold. All the muscles and skin supplied by the internal popliteal nerve, that is to say the gastrocnemius and all the flexors of the toes and ankles, will be unaffected. It is a curious fact that the external popliteal nerve seems more vulnerable to injury than the internal popliteal portion of the sciatic. These two halves of the nerve, though bound together in one sheath to form the sciatic nerve, are really distinct from one another, and indeed sometimes are separated from one another as high as the buttock, the pyriformis muscle then passing between them.

Pressure upon the sciatic nerve causing incomplete paralysis, and partial injuries of the nerve in the buttock or thigh, as from gunshot wounds, &c., usually cause more pronounced foot-drop from external popliteal paralysis than damage to the internal popliteal. This vulnerability of the external popliteal portion is also seen in the action of toxins upon the nerve, as for instance in diabetic sciatic neuritis, in which foot-drop from peroneal paralysis may be the most pronounced feature. This liability to extensor rather than flexor paralysis is seen also in most forms of multiple neuritis.

Sciatica

Sciatica, or sciatic perineuritis, though usually the consequence of some blood state or infective process such as rheumatism, gout, or typhoid fever, chills, or exposure to cold, is also sometimes produced as the result of violent muscular effort or sudden strain. I have thus seen it in a man of middle age follow immediately upon playing football, a game in which he had been induced to join, though perfectly fit and well, but yet untrained for the game. In another case an officer was blown to the ground by the explosion of a heavy shell which burst quite near to him. He was dazed by the concussion, and when he came to himself found he must have crawled to his dug-out some hundred or more yards away. On regaining consciousness he found he had sharp pain in the left buttock, which developed within a few hours into a typical sciatica, the pain rapidly spreading down the back of the thigh and into the leg and foot. Two months later, when first I saw him, he was still hobbling about on two crutches, and unable to get his heel to the ground. Injection of the nerve at the sciatic notch with novocain and saline brought about an immediate cure.

A coal-miner, to save himself being run over by a wagon running down a sloping pathway underground, was obliged suddenly to hurl himself sideways, falling on a heap of broken coal. When he tried to get up he was unable to do so on account of the severe pain in the back of the hip, which soon developed into severe sciatica, causing him to limp in a crouching attitude, in which condition I saw him three months later. In this case also saline injection brought about an immediate cure.

The mode of origin of these cases is suggestive of rupture of some muscle fibres, causing local pain, the damage rapidly setting up an acute fibrositis, spreading to involve the sheath of the neighbouring sciatic nerve. Unlike the sciatic perineuritis resulting from rheumatism and other infections, the damage from muscle strain is purely local, and affects the nerve in only one spot, usually in the neighbourhood of the sciatic notch, though the pain and disability may be apparently as great as in any severe case of sciatic neuritis of rheumatic or similar origin. In the earlier stages muscular wasting is not seen, though some emaciation of the limb from disuse will appear if the trouble continues for months. Occasionally, however, atrophy of certain groups of muscles may be seen, and in the case of the officer who had been persuaded into playing football with his men there was intense atrophy, with the loss of faradic excitability, of the gluteus maximus. This symptom showed that the small sciatic nerve was also involved, and the wasting rapidly improved after the pain had been relieved by a massive saline injection, and six weeks later the muscle had obviously increased in size, and its faradic excitability had returned.

These cases of traumatic sciatica respond extraordinarily well to treatment by massive saline and novocain injections of the nerve at the sciatic notch. Four ounces of freshly prepared normal saline should be boiled and then allowed to cool to a temperature of about 100° F. The patient lies partly over on his face, with the affected side uppermost and the hip slightly flexed. The line of the nerve is then drawn on the skin with a marking-pencil, the bony landmarks of the tuber ischii and of the trochanter being specially indicated, and the line of the nerve passes almost midway between these two points, and then curves inwards slightly to a point on a level with the top of the intergluteal fold. A fine hollow needle four inches in length is used, made of drawn steel, that will spring back into shape after bending, and fitted with a mount adapted for use with a 10 c.c. syringe such as the ‘ Record ’. After sterilization of the skin with iodine in spirit, and applying a local anaesthetic to the skin, this needle is then plunged in the line of the nerve almost vertically in order to strike the nerve near its exit from the notch. This will be found at a depth of 3¼ to 3½ inches usually, the indication that the nerve has been hit being a twinge like an electric sensation being felt in the heel or foot. Two c.c. of sterilized novocain are then injected into the nerve, taking care to hold the needle quite still, and then, using a 10 c.c. syringe, the warm saline solution is injected until from 80 to 100 c.c. have been pushed into the nerve sheath. This would be an intensely painful procedure were it not for the preliminary injection of the nerve with novocain solution. The immediate effect of the injection is to make the limb feel swollen and hot, and, if the novocain was properly placed within the nerve, anæsthesia of the foot and leg and the back of the thigh will be produced, together with paresis of the movements of the foot at the ankle. These signs will clear up in from twenty minutes to an hour at most. The patient may complain of a certain amount of stiffness and discomfort for the first night after the saline injection, and it will be advisable for him to stay in bed for twenty-four hours after being injected. On the next day the patient usually feels that the limb is much freer and that the pain and stiffness is quite gone or much better. Walking may now be possible without any limp, and the remaining pain and stiffness may now be treated by massage and daily brine baths, such as are employed at Droitwich.

In some of these cases there are additional points of intense tenderness away from the sciatic nerve, but aggravated by standing or any movements. If these points of tenderness are constant they may sometimes instantly be relieved by local injection of novocain followed by 90 per cent alcohol, and several such patches of fibrositis may be found in the neighbourhood of the posterior superior spine of the ilium, or the top of the sacrum, or between the trochanter and the crest of the ilium. Great care must be taken not to inject the trunk of the sciatic nerve with alcohol, on account of the instant motor paralysis that would ensue, requiring from twelve to eighteen months for recovery. If injection treatment be decided against, and more ordinary means of treatment are preferred, ionization with salicylate of soda, and anodyne liniments, such as equal parts of aconite, belladonna, and chloroform liniments, or iodex ointment, or menthol and methyl salicylate ointment, may be used.

Radiant heat, the light from an arc lamp, or the penetrating heat from that form of high frequency current treatment known as diathermy, may be usefully employed if ionization fails to give relief, and later deep vibratory massage will complete the cure. X-ray treatment has also produced good results.

PART II

NERVOUS SHOCK FOLLOWING CEREBRAL INJURY

Gross injuries to the brain as the result of laceration, hæmorrhage, contusion, fractured base, &c., in addition to the loss of function of localized portions of the brain from material damage done, such as hemiplegia, hemianopia, aphasia, &c., from lesions of the motor area, occipital cortex, left superior temporosphenoidal, and Broca's convolutions respectively, may produce serious alterations of the cerebral functions as a whole. These may be ascribed partly to the result of the general concussion of the cerebral cortex, and partly to reflex inhibitory trophic effects upon parts of the cortex, particularly the frontal region, produced by the gross injury or laceration of other portions of the brain. Neurasthenia, hysteria, and psychasthenia will be specially likely to develop after cerebral injury in individuals with a family history of nervous disorders, and especially of insanity, and nervous sequelæ of all kinds are more prone to develop if the patient has been given to the taking of alcohol in more than a very moderate quantity, or, worse still, to the more potent drugs such as morphia or cocaine. Again, if the patient has suffered for years from chronic insomnia, a cerebral injury is more likely to be followed by acute nervous phenomena, such as grave neurasthenia or chronic insanity. It is difficult to draw the line between the alteration of mental functions which may properly be looked upon as a natural sequel of a grave cerebral injury, and a superadded neurasthenia. The word 'traumatic neurasthenia' has been coined, and has been somewhat widely employed to cover both conditions. The term has consequently become somewhat abused, in more senses than one.

Persistent headache is one of the commonest symptoms following fractured base and other grave injuries to the brain, and may be in part due to a chronic meningitis which has been produced. The pain is usually at the back of the head and across the forehead, and is aggravated by stooping, and especially by taking any form of alcohol. Mental exertion, worry, and anxiety will aggravate such a headache and will tend to produce insomnia. This form of headache may be distinguished from neurasthenic headache often by the character of the pain in the latter form being usually described as a vertical pressure on the top of the head, which may resemble the driving in of a nail, the so-called *clavus hystericus*.

Generally, too, the manner of the patient in neurasthenia will suggest that the pain is not so excruciating as might appear from his complaints. Headaches following cerebral injuries may persist for some months, or even for years, but they are not necessarily present even when other symptoms may be obvious. Lack of mental concentration, with a diminution in the powers of endurance of mental work, with a general loss of energy and a diminished capacity for producing the best quality of work, are important symptoms that not infrequently follow grave cerebral injuries. There is often a loss of self-reliance, with nervousness and inability to make up the mind in any crisis, and even the considered judgement may be faulty. Irritability of temper, peevishness, and alterations of character are not infrequently met with, and a person who was previously pleasant and easy to get on with may become short of temper, almost insanely jealous, and liable to depreciate anything and everybody around him. Such symptoms are more liable to occur if the period of complete rest after the injury has been unduly short. After a severe cerebral injury, such as a bad concussion or fractured base, it is most important to keep the patient perfectly quiet in a dark room for a month at least, and no mental work should be undertaken for another six months. Drug treatment may be necessary to induce sleep and rest, bromides being as a rule the most helpful, though if insomnia be a prominent symptom, chloralamide or veronal may be necessary. In many cases opium cannot be dispensed with, and in obstinate insomnia, with threatening delirium and general tremor, hypodermic injections of ¼ gr. of morphia should be given every four hours until the symptoms subside. The regular administration of chloral should be avoided, owing partly to its depressant action upon the heart, and also to the fact that acute mental symptoms may follow the prolonged administration of chloral. Massage in the evening, if not too energetic, may help to induce sleep, or if the excitement is great and delirium is threatening, a hot pack, or, better still, keeping the patient continually in a warm bath at a temperature of 95°, is useful.

Sometimes, in spite of early treatment by sedatives, the delirium increases and mania in some form supervenes. If the pulse is rapid and tremor of the tongue and lips and hands is great, the danger of physical collapse and death is not remote. Continual bath treatment is one of the best remedies for this condition, combined with regular feeding with milk and eggs and soups, with the sedative treatment already described. Bad symptoms making the prognosis more grave will be thick furring of the tongue, rapid, weak, and running pulse, tremors of the hands and face, and general muscular prostration. Alcohol must never be given in these cases.

Instead of the more acute form of delirium just described, an insanity of the manic-depressive type may supervene. Excitement is less intense than in the previous form, the pulse may not be notably increased, and the tremors of the face and limbs will be absent. In this form hallucinations are less common, though delusions will be present, taking the form of having committed the unpardonable sin, and the fear of being punished, of the police, &c.

Hallucinations of sight and hearing are serious symptoms, though not necessarily of the utmost gravity, the visual hallucinations being usually confused and transient, and not taking the form of rats and spiders, snakes, &c., as are common in delirium tremens. They soon become depressed, weeping bitterly, and ultimately subsiding into a semi-stuporous state of melancholia, which may be continued for some months, and even years, before recovery takes place, though ultimately almost perfect recovery may ensue. Such patients usually require treatment in an asylum under certificates, and as a rule no drug treatment is specially indicated, though the danger of suicide must always be borne in mind.

General Paralysis of the Insane sometimes follows directly after severe blows upon the head, and is no doubt directly attributable to such injury, though the additional factor of previous syphilis is necessary for the development of this disease. The recognition of this disease in its early stages is often extremely difficult, and many mistakes have been made, and will no doubt continue to be made, in confounding this disease with neurasthenia, epilepsy, and cerebral tumour. The initial symptoms may be either physical or psychical: that is to say, the first signs may be diplopia or commencing optic atrophy, or epileptic fits, or, on the other hand, psychical phenomena such as excessive buoyancy of manner, delusions, or alteration of behaviour may first attract attention. The Wassermann reaction, both of the blood and cerebro-spinal fluid, is usually strongly positive, and the cerebro-spinal fluid obtained by lumbar puncture will also show well-marked lymphocytosis, and the presence of globulin proteid, after diluting it with an equal quantity of saturated solution of ammonium sulphate.

One of the most important points in the diagnosis of general paralysis is the manner of speech and the articulation. The latter is frequently altered in the early stages of the disease, a tendency being noticed to clip the ends of the words, the general resemblance to the speech of a semi-drunken man being fairly close. The application of certain test words and phrases will help to bring out this difficulty with the elision of the end syllables, such as ‘ Royal Irish Constabulary ’, ‘ Biblical criticism ’, &c. In addition to the resemblance of the general paralytic utterance to that of an alcoholic, it will also have to be distinguished from the defective forms of articulation also found in disseminated sclerosis, Friedreich's disease, and bulbar paralysis. An irresponsible manner of speech may betray the commencing deterioration, and commencing delusions may be noticeable, the patient's ideas being grandiose about his attributes and possessions, his physical and mental powers, and his ability to confer gifts and benefits upon all and sundry. Tremor of the facial muscles and of the tongue is often present at this stage, the tremor and over-action of the muscles of the face being especially noticeable on performing some action such as screwing up the eyes or showing the gums. In other cases the articulation is not affected, but the patient is inordinately loquacious, and a nuisance to everybody around by his constant talking and interference. The pupils may be of the Argyll-Robertson type, often unequal in size and irregular in their outline. This symptom is not always present at the commencement of other symptoms, though the loss of light reaction may appear gradually during the development of the disease. One of the early symptoms is inability to perform the usual day's work as easily and as well as usual, and for many weeks or months before the disease is recognized, the patient's office work will be complained of by his fellow workers or superiors as increasingly inefficient. With this a certain degree of physical weakness and languidness is often noticed. In many cases delusions of grandeur are absent throughout, the patient instead being depressed and morose, perhaps insanely jealous or suspicious, and a violent outbreak of homicidal mania may appear with very little warning, and perhaps be followed by violent convulsions and coma. Restraint under certificates, preferably in an asylum, is the only method of dealing with such cases. There is no treatment known which modifies or arrests the progress of a case of general paralysis of the insane. Though it has been demonstrated that the spirochætes of syphilis can usually be found in the cerebral substance of the frontal lobes, yet anti-syphilitic treatment, whether by mercury and iodide, salvarsan injections, or salvarsan auto-serum, has hitherto proved a failure.

Neurasthenia

Neurasthenic and hysterical symptoms are a common sequel to blows upon the head, though they are by no means uncommon also after other forms of accident in which the nervous system has escaped gross injury, or again they may follow periods of mental strain and exhaustion in which no physical injury has been received. The patient will complain of vague pains about the back of his head and forehead, often of a sensation of vertical pressure upon the top of his head, or again as if the top of the head were opening and shutting. He feels exhausted physically, and is unable to do light work without great mental depression and physical collapse; his lips may be tremulous, and slight twitches of the facial muscles may be noticed when questioned closely about his complaint; the hands may be very shaky, and the pulse is usually rapid. Indeed, this persistent raising of the pulse rate, or tachycardia, may be the only objective sign which it is possible to say has not been due to simulation or malingering. The deep reflexes are often unduly brisk, though the plantar reflexes may be either strongly flexor, or may even be absent.

The pupils will be normal and there will be no diplopia or any form of ocular paralysis. The optic disks should be examined in order to exclude the presence of optic neuritis or atrophy of the disks. Lack of power of mental concentration and restlessness are common features, and the patient will be unable to persist in any work that he takes up, owing to feelings of increasing mental and physical exhaustion. Vertigo is not uncommon, and tinnitus or noises in the ears of hissing or singing character are also frequent. Profuse perspirations and flushings, alternating with coldness and clamminess of the extremities, are a common complaint. Palpitations and shortness of breath after slight exertion, and a general feeling of flabbiness and inability to do any work, also are usually to be noted.

Insomnia is common, and bad dreams, especially in the earlier weeks after the injury, are sometimes troublesome, the dreams being usually terrifying, though not necessarily connected with the incident of the actual injury. In other cases of neurasthenia sleep may be excessively profound and heavy, the patient waking unrefreshed in the morning. It is almost the rule to find an excessive nervousness connected with the actual incidents of the original injury. Thus a man who has been badly thrown from a horse or injured in a carriage accident may betray an excessive nervousness in dealing with horses afterwards, and may be unable to sit with comfort in any form of carriage. The noise of a railway train may cause the victim of a railway accident to burst out into perspirations and tremors, or the noise of water may be unbearable to a person who has been rescued from a collision at sea.

A ship's engineer who sustained a bad fall when cleaning his machinery was afterwards quite unable to move about amongst his machinery when it was in motion, which he had previously been able to do without any difficulty. Usually these patients betray other symptoms of persistent neurasthenia such as tachycardia and general muscular lassitude, and the particular phobia such as described above will be unlikely to persist as an isolated symptom unless there is a history of mental instability in the family.

The Treatment of Neurasthenia, broadly speaking, will consist of prolonged rest, both physical and mental, though modifications will be necessary in individual cases. Daily general massage is often useful, though its effects must be carefully watched, as certain individuals cannot bear the manipulation for various reasons, owing to hyperæsthesia of certain parts. Isolation from friends and visitors is necessary in a number of cases, but by no means in all cases of neurasthenia. It is more particularly of value in cases of insomnia, with excessive irritability to noise of all kinds, and particularly it is useful for those individuals who become worked up and tremulous, with palpitations and perspirations, from nervousness when talking to others outside their own family circle. Often in neurasthenia, moreover, this irritability is extended to intercourse with the family circle itself, and isolation from relatives and friends is necessary as well as from casual acquaintances. The treatment must then be carried out in a nursing home or institution, and not at the patient's own house, and not only must friends and visitors be excluded, but as a rule all correspondence and the receipt of letters must be interdicted. Generally newspapers, books, and magazines may be allowed, though care must be exercised in modifying this rule in individual cases. For example, in dealing with patients who have developed neurasthenia through the war, either through stress of fighting, the result of shell explosions or the like, or while living in this country have become neurasthenic through dread of injury to relatives or husbands fighting abroad, it would be unwise to allow any daily or weekly newspapers during treatment, yet at the same time it would be highly unwise, in the case of individuals excessively overwrought with anxiety for friends abroad, to withhold from them all information. It will usually be wise to allow the receipt of letters from them in such cases, though they must not be allowed to write themselves, but merely to send messages through the nurse or doctor attending. Much patience and common sense will be required in the management of patients undergoing isolation treatment. As a rule a daily visit from the doctor is advisable, as it is largely by impressing his personality upon them and coaxing them out of their various phobias and complaints that a cure will be effected. Equally important will be the choice of a sensible and efficient nurse to be with the patient almost constantly by day. Two qualities are especially valuable in the nurse for such a case of neurasthenia, firmness and sympathy. She must be capable of being relied on implicitly by the doctor to carry out the treatment ordered, and her sympathetic attitude must not be extended into a defensive alliance with the patient against the doctor. Such an antagonistic atmosphere the doctor cannot possibly prevail against, and the only remedy then is an immediate change of nurse. Drug treatment will be of help for the combating of certain symptoms. Sodium bromide in ten- or fifteen-grain doses three times a day is valuable in the variety of neurasthenia showing irritability and insomnia, though it should be avoided in cases with depression, and when sleep is either unaffected or more profound than usual. With the bromide some preparation of valerian may occasionally be usefully combined. Strychnine is as a rule better avoided, though in the convalescent stage Easton's syrup and arsenic will be a useful tonic. Glycero-phosphates, either in medicine form or combined with casein in the form of Sanatogen, have had a great vogue, though I have not been much impressed by their value. Similarly, lymphoid serum has been widely used, both orally and by hypodermic injections. Many profess to have seen great benefit from its use. Certainly it is expensive, and this latter point is, perhaps, not unconnected with its beneficial effect by suggestion. The so-called neurasthenic serums composed of solution of sodium cacodylate have been widely used on the Continent by hypodermic injections, and there is no doubt that the daily administration of a mysterious drug by hypodermic injection may have a powerful accessory suggestive effect in promoting a cure. That is, I believe, the main value of such various treatments.

Sleeplessness may require the use of hypnotics before the habit of sleep is re-established, but their use must be carefully regulated and discretion exercised in their choice. The patient should not be allowed to know, under any circumstances, what drugs are used for this purpose, and in order to safeguard himself in this particular the doctor should himself send the prescription to the chemist, without informing the nurse what he is ordering, at the same time telling the chemist to retain the prescription. Chloralamide, or, as it should now be called, ‘ chloral formamide ’, in half-drachm doses, with thirty to forty grains of sodium bromide, is a safe hypnotic, and may be repeated nightly five or six times without harm, though it is best to order the dose to be diminished slightly every evening if sleep has been successfully produced. Veronal, especially in combination with sodium, its more soluble salt, in doses of five to eight grains, is also most valuable on occasions. This drug has lately acquired an undeserved reputation for being dangerous, on account of several deaths from suicide from overdosing with it. Morphia should never be used in such cases for the treatment of insomnia, except in cases of extreme urgency where other methods have all failed. Hyoscine in combination with bromide in doses of $\frac{1}{144}$ of a grain three times a day is sometimes of use in reducing excitability, though it is not a drug which should be continued for long in neurasthenia, although in paralysis agitans it may often be continued for many months with apparent advantage.

Prolonged immersion in warm baths is another valuable means of inducing calm in cases of continued excitability, restlessness, and palpitation. This form of treatment is more particularly useful when mental symptoms are prominent, and it is frequently used in asylums for such cases. In slighter cases of excitability with insomnia, facial and head massage may act as a useful sedative, and when it has this good effect this treatment is preferable to drugs. **Diet** must be nutritious and easily digested. Milk, when it can be taken, should be given up to three or four pints a day, with, in addition, light meals of fish, egg, custard, porridge, &c., ordinary meals with meat and vegetables being added when the convalescent stage is reached. Malt foods, of which there are numerous types upon the market, are often a useful addition, and an important point in the feeding up of these patients is absolute regularity in the administration of the food. The patients should be weighed at the commencement of the treatment, and every week afterwards, though the facts of the weight should be concealed rigidly from them. This is an important point, as often weight is lost in the first week or two, which may cause the patients to worry unduly if the fact is announced to them. If it can be arranged, therefore, the doctor should do the weighing himself, as otherwise it is difficult to ensure the weight being kept secret, even though the nurse be trustworthy. Whether weight is gained or lost, the patient must be encouraged and told that all is going on well, as it should be borne in mind that suggestion is the key-note of all treatment in neurasthenia.

Treatment by Suggestion and Psycho-analysis.—In obstinate cases of neurasthenia all treatment by isolation, massage, electricity, and drugs sometimes fails, and treatment by hypnotic suggestion may be of value. I use the term advisably, though practitioners who make a speciality of this line of treatment usually disclaim the employment of actual hypnosis, and the method is now called **passive suggestion**. Psychoanalytic methods are practically not required in the treatment of neurasthenia and kindred mental phenomena, due to recent injuries and shocks of different kinds. The essential point of this treatment is that it searches out and brings to light the hidden psychical trauma and its buried complexes acquired years before, and whose origin has been lost and perhaps completely forgotten. For a description of this method books and writings on psychiatry should be consulted (cf. Stoddart, *Review of Neurology and Psychiatry*, June 1915).

Psychasthenia is a nervous condition in which mental phenomena predominate over physical, whereas in neurasthenia physical signs, such as tremor, palpitations, insomnia, and loss of weight, are prominent features.

Obsessions are a common complaint in psychasthenia, and various phobias, and especially dread of becoming insane, are almost constant. Although psychasthenia, previously more or less latent, may be aggravated after injuries and shock, yet it more strictly belongs to the domain of the alienist, as its ætiology depends upon congenital mental instability and neuropathic heredity.

Nervous exhaustion is perhaps as good a term as any to apply to the temporary neurasthenic phenomena, states of nervous collapse, and amnesia that have been such a common feature in patients invalided during the present war in France. A soldier previously healthy, after being exposed for days and nights to continual harassing noises and shell and rifle fire, possibly with insufficient food and certainly with great insufficiency of sleep, is liable to lose his nerve, becoming temporarily irresponsible and apparently a coward. It is particularly the sound of heavy gun firing and the noise of shells that gives rise to irritability, rifle firing being taken no notice of, but the moment the guns start firing, especially at night, the man gets restless and walks up and down the trench continually, while the firing lasts, a mental condition which has become known as 'Shell-Fever'. In a half-dazed condition he may desert his post, or if sent back to the base on account of symptoms of excessive nervousness being noticed, he may fall asleep for three or four days, being waked up for food at intervals and immediately falling asleep again. In many of the lighter cases sleep, followed by a few days' light duty at the base, may be sufficient to restore his mental balance, and his courage and his physical strength soon return to the normal. In other cases, bad dreams, in which they imagine themselves pursued by Germans, with ordinary symptoms of neurasthenia, such as tremors and palpitations, may persist for even days and weeks. Such patients are apt to be highly emotional, and may break down into tears if asked to describe their experiences at the Front. This is especially the case if the man's regiment has been severely handled, and numbers of his comrades and brother officers have been killed. These cases require more prolonged treatment, being sent home to England, and may sometimes be treated with advantage by isolation and overfeeding for a few weeks on the lines above described, followed by a month's rest at home, living an open-air and out-of-door life. Usually these cases do well, their nervousness and tendency to break down gradually improves, and their general sense of well-being and courage may return to the normal, with the wish to return again to the fighting line. The worst cases are those whose heredity is bad, with neurasthenia, epilepsy, and insanity in near relations. Some of these will not get well, and neurasthenia will remain permanent, and symptoms of psychasthenia or even insanity may appear. A common case of the development of acute nervous collapse is of soldiers being buried by the explosion of a shell near by, blowing in the trench, and all degrees of nervous collapse with complete loss of memory may result. Such a patient may present total loss of memory, and every fact of his life preceding the accident may be strange to him. He may not know his name, occupation, or where he lived, what unit he belonged to in the army, or in what country he happens to be. Such a condition of total amnesia is liable to be mistaken for malingering, though careful testing and cross-examination will show the man to be perfectly consistent in his replies. Such patients quickly learn again to read and write, and soon pick up past facts of their career, though the true recovery of memory may be long delayed. Such a patient, if placed under hypnosis, may in this state remember perfectly all the facts of his life which in his waking state are forgotten, and indeed a state of double consciousness may be set up in which the patient suddenly passes from a normal individual into a state of amnesia, in which his life began at the point of recovery of consciousness following the accident. Such a prolonged state of double consciousness is unlikely to be met with except in an individual with neuropathic heredity.

Prolonged work alone amongst dangerous conditions, such as a telephone operator in the field, or a sharpshooter, or on an observation post, may also cause a man to suffer from nervous collapse, the symptoms commencing with irritability and difficulty in passing on messages at normal speed, followed by complete inability to understand or receive messages, and increasing fear of his exposed position near the fighting line. These are usually not serious cases, recovering with rest and sleep and regular food amongst the peaceful surroundings of a base hospital, particularly in England. The shock of a large high-explosive shell bursting close by may cause complete unconsciousness without burying the patient, and the recovery of consciousness may be gradual, tactile sensation returning while yet complete deafness and blindness may be present. This exhaustion of the special senses is of course cerebral in origin, and not due to any lesion or exhaustion of the peripheral end organs, while the pupils will react to light though blindness is complained of. The power of vision and hearing will probably return within an hour or so, though complete inability to move the body or limbs may continue for days.

Under treatment by rest the power usually returns gradually, and ultimately the patient may make a complete recovery, either in a few days, or it may take several weeks or even months, according to his nervous temperament and his mental stamina. Such temporary paralysis of the whole body and limbs may leave behind any of the known forms of hysterical paralysis or contracture. Thus a patient now under treatment after such an experience has recovered apparently completely with the exception of hysterical contracture of the right foot, without any anæsthesia. Such a condition must be treated on the lines described under hysterical paralysis.

Hysteria

Objective symptoms of **hysteria** may be combined with the symptoms of neurasthenia above described, though not infrequently they may appear alone. Such are hemiplegia, or monoplegia, paraplegia, hemianæsthesia, patchy anæsthesia, amblyopia with contraction of the visual fields and sometimes actual complete blindness. Hysterical convulsions may occur which are sometimes difficult to distinguish from epileptic fits. Hysterical hemiplegia will be distinguished from paralysis of organic origin by the following signs. There will be no weakness of the lower half of the face on the paralysed side, the position and type of rigidity of the paralysed arm will be characteristic, not being flexed at the elbow to a right angle, and the fingers being usually straight with the thumb adducted to the forefinger, instead of the fingers being flexed with the thumb bent inside as in organic hemiplegia. The gait in the hysterical form is not very unlike the organic type, the foot being dragged. The deep reflexes may be normal, or slightly increased, though the difference, if any, on the two sides will be slight ; usually the deep reflexes are equal on the two sides. Of the superficial reflexes the abdominal and epigastric reflexes will be equal on the two sides, and there will be no extensor plantar reflex, the response being of normal flexor type, or else absent altogether on the paralysed side, especially if there is hemianæsthesia also.

All forms of curious gait are sometimes met with in hysterical paresis, gross tremors of one or both legs and curious prancing movements of a type not to be reconciled with any known organic lesion, but the recognition of these curious forms of gait as being of functional origin is to be acquired only by experience.

Hemianæsthesia to all forms of sensation is a frequent symptom accompanying hysterical paralysis, though it may occur without any weakness of the limbs, and conversely, hysterical hemiplegia or paraplegia may occur without any anæsthesia being present. The loss of sensation on one side may be complete or partial both in extent and in degree. Sometimes the borders limiting the anæsthesia do not correspond to any possible anatomical lesion, when hysteria as the cause may be diagnosed by exclusion. Thus I have seen hysterical hemianæsthesia involve one-half of the head and neck, arm, and trunk down to the level of the navel, below which point sensation was normal. It is inconceivable to imagine a possible anatomical lesion producing such a distribution of the anæsthesia. In other cases I have seen hysterical anæsthesia extend for more than an inch across the middle line, which again is anatomically impossible. The reflexes will be unaltered, the abdominal and epigastric reflexes being as brisk as on the other side. Sometimes the anæsthesia is patchy, the shape and distribution of the anæsthetic areas being apparently without reason. Accompanying hysterical hemianæsthesia there is sometimes found affection of the special senses on that side; the visual fields may be constricted, particularly that of the eye on the hemianæsthetic side, and the visual acuity will also be found to be diminished in that eye, and possibly recognition of colours with that eye will be lost. In addition to the visual defects there may be unilateral loss of smell, taste, and hearing on the same side as the hemianæsthesia. The plantar reflex may be lost on the anæsthetic side, though there may be no motor weakness. The loss of the sense of position of the anæsthetic limb will be apparently complete, though he may at once be able to find the different parts asked for with the other hand, while in organic lesions producing anæsthesia there will be considerable difficulty in finding the anæsthetic hand with the sound one. This latter symptom, or astereognosis, is particularly seen in extensive lesions of the posterior roots, posterior columns, or of the region of the optic thalamus.

The only organic lesion likely to affect vision, in addition to producing hemianæsthesia, with unilateral diminution of smell, taste, and hearing, would be destruction of the optic radiations at the back of the internal capsule, causing hemianopia and not concentric contraction of the fields of vision. In all probability, in these cases of organic origin there will also be some sign of hemiplegia, the leg being probably more paralysed than the arm, and there will be increase of the knee and Achilles jerks on that side, with extensor plantar reflex.

In hysterical anæsthesia it will be noticed that the conjunctival reflex of the anæsthetic eye will be undiminished in its sensitiveness, a useful point in the differential diagnosis from organic lesions.

Occasionally the visual fields of both eyes are very much contracted, the so-called pin-point fields. If the visual acuity of the other eye is normal, and if the diminished acuity of vision on the affected side can be shown not to be due to an error of refraction, or not improved by correcting with glasses any error that is present, it can easily be demonstrated that the diminished power of vision is not of organic origin, but is either hysterical or due to malingering. This may be done by various tests, such as by placing a strong prism in front of the good eye, base downwards, thus producing vertical diplopia, and then telling the patient to read first the upper and then the lower sets of test letters.

The patient will not know to which eye each image belongs, though the observer knows that the false image, when using the prism, is projected in the direction of its apex. Consequently, if the apex is uppermost, the upper image will belong to the eye using the prism, and the lower image to the other eye. Usually the patient will read each set of letters from the two images equally distinctly, thus proving an inconsistency in the visual acuity, and therefore a functional origin for the amblyopia. Sometimes the amblyopia amounts to total loss of vision, the patient even declaring that he cannot see light with one or both eyes. The pupil reflex to light in these cases will be unaffected, unless atropine drops or belladonna is being fraudulently used, and if only one eye is complained of, its visual acuity can be demonstrated to be normal by the prism or other similar test. The difficulty is to distinguish hysterical amblyopia from malingering, as these tests just described do not separate them.

Hysterical amblyopia is most likely to be mistaken for the loss of vision produced by retrobulbar neuritis, particularly that form of retrobulbar neuritis occurring in early disseminated sclerosis. The difference in the pupil reaction, however, will at once distinguish hysterical amblyopia from the organic condition, as the pupil reaction to direct light stimulus in retrobulbar neuritis will be diminished in proportion to the degree of amblyopia, whereas in hysterical amblyopia the pupil reaction will be normally brisk. If one eye only is affected with retrobulbar neuritis, as is commonly the case, the pupil reaction of the affected eye will be sluggish to direct light, though its consensual reaction will be brisk when light is thrown upon the other pupil.

Hysterical Monoplegia

Either limb may be involved alone by paresis in hysteria. When this is the case the limb is usually flaccid, with only slight contracture of the hand when the upper limb is involved. Sometimes the limb is anæsthetic, in the case of the upper extremity the anæsthesia spreading over the shoulder perhaps to the middle line in front and behind, involving the fore quarter. This anæsthesia may be apparently complete to all forms, including sense of position, and occasionally there is also found a form of plastic rigidity, known as flexibilitas cerea, a condition of local catalepsy, in which the limb will remain fixed and motionless for a considerable time in any position in which it may be placed. This sign is pathognomonic of hysteria, being never met with in organic lesions. In other cases in which there may be total hysterical paralysis of the upper extremity there may be no anæsthesia at all. Hysterical paraplegia is comparatively rare in adults, though it is not so uncommon in children and young women. Usually the paraplegia is of the flaccid type, and there may be no anæsthesia, or there may be anæsthesia of the stocking type involving the feet and legs to just above the knees.

The deep reflexes will be normal as a rule, though occasionally bilateral rectus clonus may be obtained, and even a form of ankle clonus, which latter is often very difficult to distinguish from true ankle clonus. It is, however, less consistent and more spasmodic, being violent at times, and the next minute perhaps unobtainable. The plantar reflexes are either flexor or else absent altogether. The sphincters will be unaffected.

The differential diagnosis of functional paraplegia will be necessary from spastic paraplegia due to disseminated sclerosis, spinal caries, and other organic lesions of the spinal cord; from severe bilateral anterior poliomyelitis; from multiple neuritis, and lastly from malingering. Usually spastic paralysis due to organic lesions is fairly easy to distinguish. In these cases adductor spasm is usually present, so that if one leg is pulled aside by catching hold of the ankle, the other leg follows immediately, owing to tonic rigidity fixing the lower extremities to the pelvis. The briskness of the knee-jerks and a regularly obtained ankle clonus, with extensor plantar reflexes, will at once mark the organic nature of the case, whereas anæsthesia, if present, will usually extend above the lower extremities to some level of the trunk, such as the navel, when the lesion is at the tenth dorsal segment. In poliomyelitis the paraplegia will be quite flaccid and hypotonic, and there will be no trace of anæsthesia, though hyperæsthesia is sometimes present. In this disease the rapid onset of muscular wasting, with reaction of degeneration on electrical testing, demonstrates the existence of an acute lower motor neurone lesion, and the deep reflexes will usually be absent.

Paralysis of the lower extremities due to multiple neuritis may be distinguished in the early stages by a flaccid paralysis, loss of the knee-jerks and Achilles jerks, and perhaps bilateral foot-drop. It is true that in the early stages of some forms of multiple neuritis, such as the alcoholic and the post-diphtheritic, the knee-jerks may be temporarily brisk, though after two or three weeks almost invariably they become lost.

Foot-drop is not always present in the slighter forms of multiple neuritis, when the weakness of the legs amounts to only slight loss of power and unsteadiness in walking, though in more severe cases the paralysis of the dorsiflexors of the ankles and toes will be pronounced, and muscular wasting with reaction of degeneration on electrical testing will definitely prove the weakness to be due to organic disease, and not of functional origin. The anæsthesia in multiple neuritis of the lower extremities is sometimes definitely of the stocking type, very similar to what is met with in hysterical paraplegia, though usually the anæsthesia fades away gradually up the leg, and does not end with a sharp margin about the level of the knee, as it often does in hysterical paralysis. The plantar reflexes may be absent in both conditions, but the deep reflexes are practically never lost in functional disease. The condition that is perhaps the most frequently mistaken for functional paraplegia is the weakness of the legs occurring in disseminated sclerosis. This is really of the spastic type, though the rigidity of the limb from muscular hypertonus may be only slightly marked. Paraplegia in disseminated sclerosis may develop almost suddenly, and there may be a history of a fall or an accident of some kind. The subject is usually a young woman in the early twenties, and very possibly of a neurotic type, so that an obscure paralysis of one or both lower extremities, coming on for no apparent or an insufficient cause, may easily lead to the diagnosis of functional or hysterical paraplegia being suggested. The progress, too, of the case may seem to confirm the diagnosis of hysteria, inasmuch as it is extremely common, indeed the rule, for cases of rapid or sudden onset of paraplegia in disseminated sclerosis to recover to a considerable extent after a few weeks. It is the examination of the reflexes, more than any other point, which will give the clue to the recognition of disseminated sclerosis. In these cases the knee-jerks and Achilles jerks are brisk, with very probably considerable exaggeration of the knee-jerk, and ankle clonus in one leg, while the plantar reflex will be of the extensor type in that leg, and perhaps on both sides. Other points indicative of organic disease in such a case will be sphincter paralysis with either retention or incontinence, loss of the abdominal reflex on one or both sides, diplopia due to paresis of the ocular muscles, or the signs of **retrobulbar neuritis.** This latter is a common symptom in early disseminated sclerosis, and its onset is frequently so acute as to be described by the patient as sudden. There will be considerable diminution of the acuity of vision, and central scotoma is frequent, for colours only in the slighter cases, though it may be almost complete for the form of objects also. The peripheral fields in these cases may remain nearly normal, though occasionally for a few days there may be complete amaurosis with loss of light-perception.

Ophthalmoscopic examination in cases of retrobulbar neuritis may be able to demonstrate slight hyperæmia of the disk, or even optic neuritis with two or three diopters of swelling. Ultimately pallor of the disk and optic atrophy are likely to supervene. The pupil reaction to light is diminished in proportion to the loss of visual acuity (see *ante*, p. 114). Another form of visual trouble in hysteria which may result from shock or injury of various kinds is **photophobia** and retinal hyperæsthesia. This form of photophobia differs from that due to an acute conjunctivitis in the absence of the evidence of conjunctival inflammation and of lachrymation. Blepharospasm is the prominent symptom, and in order to avoid bright light the patient will haunt dark rooms or insist on wearing tinted glasses. Another much rarer hysterical affection of vision is persistent spasm of the ciliary muscles, causing apparent myopia, which disappears under the daily use of atropine drops, but reappears when these are left off. Combined with this ciliary spasm may be diplopia due to persistent tonic overaction of one of the external ocular muscles, the strabismus varying at times from convergence to divergence of the eyes.

Hysterical bilateral **ptosis** is sometimes met with, and this may be recognized as such in the absence of other signs of ophthalmoplegia, the movements of the eyes remaining normal with the exception of the eye-lids, and by the fact that when the patient is urged to make strong efforts to open the eyes, the frontalis may be seen to contract in the effort to aid the levator palpebrae, though at the same time there is visible contraction of the orbicularis palpebrarum preventing the eyes from being opened. Hysterical ptosis must be carefully distinguished from the ptosis met with in myasthenia gravis. In this disease there will usually be complaints of diplopia, and transient strabismus may be noticed when the eyes are tired, or there may be complete external ophthalmoplegia. Usually, moreover, in addition to the weakness of the levator palpebræ in myasthenia, weakness of the frontalis on both sides is present, giving rise to the ‘ myasthenic facies ’, or ptosis with a smooth forehead. Other evidences of myasthenia will also be present, such as weakness of the orbicularis oris, and the rapid fatigue of certain limb and trunk muscles, weakness of chewing, loss of voice towards the end of the day, &c.

Treatment of Hysterical Paralysis and Anæsthesia

As a rule hysterical paralysis with anæsthesia may usually be quickly cured if its treatment is firmly undertaken soon after its onset. For example, a carpenter employed on a building injured his left thumb by a slight punctured wound one evening about 6 p.m. The wound bled somewhat freely, which made him rather nervous, and by the time he was having his supper, at 8 o’clock, he noticed that the little and ring fingers were becoming contracted down into the palm. By midnight the contracture of the hand had increased so much that all the fingers and the thumb were now strongly flexed into the palm. Becoming alarmed about his condition, he went at once to St. Mary's Hospital, where this condition was noted and it was found that the hand up to the wrist was also completely anæsthetic. When I saw him on the following morning at my Nerve Department, I found the thumb and all the fingers of the left hand strongly flexed, the fingers over the thumb, the contraction resisting strong efforts to pull the fingers out. All sensation to touch and to pin-prick was lost upon the hand up to the level of the wrist-joint, where the anæsthesia terminated abruptly.

The proper method of treating these cases is by means of strong faradism applied by a small electrode or a wire brush to the moistened skin, in order, by means of a powerful stimulation, to break down the psychical auto-inhibition which produces the hysterical anæsthesia : that is to say, the stimulus is made so powerful that the patient is forced to admit that he feels. As soon as this admission is gained the patient is encouraged by telling him that the sensation will rapidly improve, and that the electricity will be more and more strongly felt, and he is told that he will be cured in a few minutes, thus using the method of suggestion for the cure of the patient, both in the direction of encouragement, and by the paraphernalia of an apparently complicated noisy machine, together with a powerful and mysterious sensory stimulus. Using this method, the patient above referred to was completely cured within two or three minutes, the anæsthesia and the contracture disappearing rapidly.

When hysterical contracture and anæsthesia has been allowed to persist for many months or even years, it is often very much more difficult to cure, and may require many applications of the treatment. The personal element, and domination of the patient by the physician, is necessary in the treatment of all these cases, as it is not the electricity as a therapeutic agent which cures, but the general force of suggestion treatment. For this reason, cases of this nature may not improve if they are handed on for treatment in institutions by massage and medical electricity. The same form of treatment, with the necessary modifications that common sense will supply, may be used for the treatment of hysterical amblyopia, aphonia, tachypnoea, &c.

A good instance of a soldier sent home from France suffering from what may be called hysterical sciatica, with extreme difficulty of walking, may be cited. He got wet through in August 1914, during the retreat from Mons, and developed pains round his hips and down his right thigh. He was sent back to England, and for nine months was treated in various convalescent homes and military hospitals, having had forty baths at Droitwich, amongst other treatments. He was ultimately sent to me in May 1915, after being incapacitated for nine months, when I found him hobbling with apparently the greatest difficulty, requiring a stick to support himself on one side, leaning over to his left side and dragging his right leg in a stiff and rigid manner. The gait was obviously hysterical, and although he complained of apparent intense tenderness and hyperæsthesia of the thigh, I applied the same treatment by faradism and suggestion as described above, with the result that he was completely cured within five minutes, and was able to walk without a stick and run across the room. He appeared delighted with the result, and said he would now be able to go back to the Front, and rather pertinently asked why he could not have been cured before. I mention this in evidence that the man was not malingering, which is the one difficult point of distinction from such hysterical affections. It is comparatively easy to decide between organic disease on the one hand, and functional loss on the other, but whether the functional affection is hysterical or malingering may be difficult to determine.

Hysteria, when prolonged for months, is very apt to have malingering grafted upon it, and what was at first a purely psychical disturbance of function may ultimately be perpetuated mainly by the intention to deceive. There are no definite rules to guide one certainly in all cases, and each must be decided on its merits, the physician being careful not to be prejudiced beforehand. It is in the cases arising out of the Workmen's Compensation Act that the greatest difficulty occurs, and practitioners who are employed in the interests of large corporations, or who are frequently called by solicitors for the defence of the workman, are rarely unbiased witnesses on the vexed question of nervous injuries following accident.

INDEX

Abscess, cerebral, 28.
Achilles jerks, 52.
Alcohol injection, 50, 77.
Amblyopia, 122.
Amnesia, 106, 108.
Anæsthesia, 39.
  hysterical, 121.
  saddle, 52.
  segmental, 39.
  therm-, 42.
Analgesia, crossed, 42.
Ankle clonus, 116.
Aphasia, 29.
Aphonia, 122.
Argyll-Robertson pupils, 11, 97.
Articulation, 97.
Astereognosis, 112.
Atrophy, progressive muscular, 48.

Baths, 104.
Bedsores, 44.
Blepharospasm, 119.
Brachial plexus, 53.
Brain, concussion of, 13.
  œdema of, 15.

Catalepsy, 115.
Cauda equina, 52.
Cerebral compression, 14.
  contusion, 16.
Cervical sympathetic paralysis, 56.
  rib, 56.
Cheyne-Stokes, 15.
Ciliary spasm, 119.
Clavus hystericus, 93.
Claw hand, 68.
Collapse, nervous, 108.
Coma, 14.
Concussion of the brain, 13.
  spinal, 31.
Contracted visual fields, 113.
Contrecoup, 21.
Convulsions, 98.
  hysterical, 110.
Cord, inner, 60.
  lumbo-sacral, 52.
  outer, 60.
  posterior, 59.
  spinal lesions, 36, 46.

Deafness, nerve, 10.
Delirium, 95.
Delusions, 95, 96, 97.
Depression, 98, 99.
Diplopia, 10, 96, 119.
  crossed, 11.
Dislocation of the shoulder, 60.
Disseminated sclerosis, 118.
Double consciousness, 109.
Dreams, bad, 100, 107.

Endarteritis, gummatous, 26.
Epilepsy, 96.
  Jacksonian, 19.
Exhaustion, nervous, 106.
Exostoses, 27.

Faradism, strong, 121.
Fibrositis, acute, 88.
  chronic, 50.
  neuro-, 76.
Fracture of the base, 9.
  depressed, 15.
  dislocations, 36.

Gasserian ganglion, 11.
Gummatous endarteritis, 26.

Hæmorrhage, extra-dural, 15, 35.
  intracerebral, 15.
  intrameningeal, 17.
  — spinal, 33.
  meningeal, 15.
Hallucinations, 95.
Headache, 26, 93.
Hemianæsthesia, 19, 110.
  hysterical, 112.
Hemianopia, 18, 113.
  bi-temporal, 11, 12.
Hemianopic pupil, 12.
Hemiplegia, 110, 113.
Hyperæsthesia, 75, 123.
Hysteria, 51.
Hysterical contracture, 121, 122.
  convulsions, 110.
  gait, 123.
  hemianæsthesia, 112.

Injection, alcohol, 50, 77.
  saline, 88.
  salvarsan, 98.
Insomnia, 94, 100, 104.
Interossei, 69.
Ionization, salicyl, 73.
Irritability, 101.
Isolation, 101, 108.

Jacksonian epilepsy, 19.

Knee-jerks, 52, 79, 117.

Lumbar puncture, 24, 96.
Lumbrical muscles, 66.

Malingering, 123.
Mania, 95.
Melancholia, 96.
Migraine, ophthalmoplegic, 11.
Monoplegia, 110.
Myasthenia, 120.
Myelitis, sacral, 71.
Myositis, ischæmic, 75.

Nerve, anterior crural, 79.
  external popliteal, 86.
  fifth, 10.
  fifth cervical, 53.
  inferior gluteal, 81.
  inner cord, 60.
  long thoracic, 58.
  lumbo-sacral cord, 52.
  median, 65.
  musculo-cutaneous, 60, 65.
  musculo-spiral, 61.
  obturator, 80.
  optic, 12.
  outer cord, 60.
  posterior cord, 59.
  posterior interosseous, 62.
  posterior scapular, 58.
  sciatic, 81.
  sixth cervical, 54.
  spinal accessory, 57.
  supra-scapular, 61.
  third cranial, 10, 11.
  ulnar, 67.
Nervous collapse, 108.
  exhaustion, 106.
Neurasthenia, 96, 98.
Neuritis, ascending, 71.
  brachial, 71.
  occupation, 67.
  retrobulbar, 114, 118.
Neuroma, painful, 74.

Obsessions, 106.
Œdema of the brain, 15.
Ophthalmoplegia, 120.
Optic atrophy, 96.
  chiasma, 11.
  disk, atrophy of, 12.
Osteo-arthritis, 49.

Pain in the back, 49.

Paralysis, Brown-Séquard's, 42.
  cervical sympathetic, 56.
  cranial nerve, 10.
  of the diaphragm, 37.
  Erb's, 53.
  facial, 10.
  functional, 123.
  general, 96.
  hysterical, 62, 110.
  Klumpke's, 55.
  musculo-spiral, 61.
  posterior cord, 59.
  pressure, 85.
  sixth nerve, 10.
  spastic, 116.
  sphincter, 118.
  of the sympathetic, 38.
Paraplegia, 110.
Perspirations, 99.
Phobias, 100, 106.
Photophobia, 119.
Plexus, brachial, 53.
  lumbo-sacral, 71.
  sacral, 52.
Priapism, 37.
Ptosis, 10, 11, 119.
Psychasthenia, 106.
Psycho-analysis, 105.
Puncture, lumbar, 24, 96.
Punctured wounds, 24.
Pupil, Argyll-Robertson, 11, 97.
  hemianopic, 12.
  reaction, 114.
Pyrexia, 37.

Reflex, deep, 41.
  — Achilles jerks, 52.
  — knee-jerks, 52, 79, 117.
  superficial:
  — abdominal, 111.
Reflex, superficial (*continued*).
  — conjunctival, 113.
  — epigastric, 111.
  — extensor plantar, 31, 111, 113, 116.
  — plantar, 116.
Rhomboids, 58.

Saline injection, 88.
Salvarsan, 98.
Sciatica, 87.
Sciatic nerve, small, 81.
Sciatic paralysis, 81.
— puerperal, 85.
Segmental muscular supply, 38.
Smell, loss of, 13.
Spasm, ciliary, 119.
Strabismus, 119.
Stump, painful, 74.
Suggestion, 103, 105.
  hypnotic, 105.
  treatment, 122.
Suicide, 96.

Tabes dorsalis, 46.
Tachycardia, 99.
Tachypnœa, 122.
Thenar eminence, 66.
Thrombosis, cerebral, 30.
Tinnitus, 99.
Trapezius, 57.
Tremor, 95, 97.
Tumour, cerebral, 27, 96.

Vertigo, 99.
Visual fields, 113.

Wassermann reaction, 96.
Wire brush, 121.
Wrist, dropped, 64.

X-rays, 91.

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